Inventor Search

=> d ibib abs 1-1

L4 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:555770 HCAPLUS
DOCUMENT NUMBER: 137:90608
TITLE: Methods of diagnosis and treatment of **osteoporosis**
INVENTOR(S): **Lewandrowski, Kai-Uwe; Trantolo, Debra J.**
PATENT ASSIGNEE(S): Cambridge Scientific, Inc., USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002057795	A2	20020725	WO 2002-US1566	20020117
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002137082	A1	20020926	US 2002-54171	20020117

PRIORITY APPLN. INFO.: US 2001-263109P P 20010119
US 2001-304887P P 20010712

AB A method of detecting **osteoporosis** in a mammalian is disclosed herein which includes: (a) obtaining a sample of a bone related tissue or cells; and (b) measuring the concn. of at least a marker which is either bacteria, bacteria produced factors, or HSPs. The method may further include comparing the concn. with concns. from the same individual over a period of time or against a std. concn. The marker may be a bacteria, a chaperone mol., or a bacteria produced. The method may include a first assay and a second assay over a period for example of at least about 12 h. The concn. of HSP can be measured using an immunoassay. The assay may use a nucleotide mol. encoding HSP. Also provided herein is a method of treating or preventing **osteoporosis** caused by a bone disease which includes administering to a mammalian subject a therapeutically effective amt. of a formulation which is either an HSP antigenic formulation or a bacterial antigenic formulation. The **osteoporosis** can be caused by a bone disease induced by bone infectious agents such as viruses, bacteria, fungi, protozoa and parasites. The HSP can be further complexed with an antigenic material or formulated in combination with an adjuvant. The antigenic material can be a peptide or a protein having an antigenic determinant of a virus, bacteria, fungi, protozoa or parasite that induces a bone disease. The antigenic material includes an antigenic determinant of a virus. Further, the methods disclosed herein can be practiced using a kit formed according to the methods disclosed herein.

=> d ind

L4 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS on STN
IC ICM G01N033-68
CC 9-16 (Biochemical Methods)
Section cross-reference(s): 4, 14
ST diagnosis treatment **osteoporosis**
IT Hepatitis
(B; methods of diagnosis and treatment of **osteoporosis**)
IT Hepatitis
(C; methods of diagnosis and treatment of **osteoporosis**)
IT Heat-shock proteins
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(HSP 60; methods of diagnosis and treatment of **osteoporosis**)
IT Heat-shock proteins
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(HSP 70; methods of diagnosis and treatment of **osteoporosis**)
IT Heat-shock proteins
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(HSP 90; methods of diagnosis and treatment of **osteoporosis**)
IT Densitometry (optical)
(bone; methods of diagnosis and treatment of **osteoporosis**)
IT Proteins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(gapstatin; methods of diagnosis and treatment of **osteoporosis**
)
IT Infection
(measles; methods of diagnosis and treatment of **osteoporosis**)
IT Adenoviridae
Animal tissue
Arbovirus
Bacillus bronchiseptica
Bacteria (Eubacteria)
Biomarkers (biological responses)
Body fluid
Bone
Campylobacter rectus
Cell
Computer program
Cytomegalovirus
Databases
Diagnosis
Epitopes
Escherichia coli
Fungi
Fusobacterium nucleatum
Haemophilus actinomycetemcomitans
Haemophilus influenzae
Hantavirus
Human
Human coxsackievirus
Human echovirus
Human herpesvirus 1
Human herpesvirus 2
Human immunodeficiency virus 1
Human immunodeficiency virus 2

Human poliovirus
Immunoassay
Influenza
Mumps virus
Mycobacterium tuberculosis
Neisseria gonorrhoeae
Neisseria meningitidis
Osteoporosis
Papillomavirus
Papovaviridae
Parasite
Pasteurella multocida
Porphyromonas gingivalis
Prevotella intermedia
Probability
Protozoa
Respiratory syncytial virus
Rhinovirus
Rotavirus
Rubella virus
Salmonella
Simulation and Modeling, biological
Staphylococcus aureus
Staphylococcus epidermidis
Statistical analysis
Test kits
Virus
(methods of diagnosis and treatment of **osteoporosis**)

IT Heat-shock proteins
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(methods of diagnosis and treatment of **osteoporosis**)

IT Bone, disease
(osteopenia; methods of diagnosis and treatment of **osteoporosis**)

IT Human herpesvirus 3
(varicella from; methods of diagnosis and treatment of **osteoporosis**)

IT 37333-47-4, Dermonecrotoxin
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(methods of diagnosis and treatment of **osteoporosis**)

IT 60267-61-0, Ubiquitin
RL: ANT (Analyte); BSU (Biological study, unclassified); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(methods of diagnosis and treatment of **osteoporosis**)

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=> d que stat l29
L10       13791 SEA FILE=HCAPLUS ABB=ON  ?OSTEOPOROS? OR ?BONE?(W)?LOSS?
L12        1110 SEA FILE=HCAPLUS ABB=ON  L10 AND (?SCREENING? OR ?DIAGNOSIS?)
L13         557 SEA FILE=HCAPLUS ABB=ON  L10 AND ?SCREENING?
L15           2 SEA FILE=HCAPLUS ABB=ON  L13 AND ?ENDOTOXIN?
L19           3 SEA FILE=HCAPLUS ABB=ON  L12 AND ?ENDOTOXIN?
L20           1 SEA FILE=HCAPLUS ABB=ON  L12 AND ?GAPSTATIN?
L21           1 SEA FILE=HCAPLUS ABB=ON  L12 AND ?DERMONECROT?
L24           4 SEA FILE=HCAPLUS ABB=ON  L15 OR L19 OR L20 OR L21
L25           1 SEA FILE=HCAPLUS ABB=ON  L12 AND (S OR ?STAPHYLOCOCCUS?)(W)?AUR
                 EUS?
L26           1 SEA FILE=HCAPLUS ABB=ON  L12 AND ?BRONCHISEPTICA?
L27           1 SEA FILE=HCAPLUS ABB=ON  L12 AND ?FUSOBACTERIUM?
L28           1 SEA FILE=HCAPLUS ABB=ON  L25 OR L26 OR L27
L29           4 SEA FILE=HCAPLUS ABB=ON  L24 OR L28

=> d ibib abs l29 1-4

L29  ANSWER 1 OF 4  HCAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2002:555770  HCAPLUS
DOCUMENT NUMBER:          137:90608
TITLE:                    Methods of diagnosis and treatment of
                          osteoporosis
INVENTOR(S):              Lewandrowski, Kai-Uwe; Trantolo, Debra J.
PATENT ASSIGNEE(S):       Cambridge Scientific, Inc., USA
SOURCE:                   PCT Int. Appl., 57 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2002057795      A2   20020725       WO 2002-US1566   20020117
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
             RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ,
             VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
             CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2002137082      A1   20020926       US 2002-54171    20020117
PRIORITY APPLN. INFO.:                   US 2001-263109P  P  20010119
                                         US 2001-304887P  P  20010712
```

AB A method of detecting **osteoporosis** in a mammalian is disclosed herein which includes: (a) obtaining a sample of a bone related tissue or cells; and (b) measuring the concn. of at least a marker which is either bacteria, bacteria produced factors, or HSPs. The method may further include comparing the concn. with concns. from the same individual over a period of time or against a std. concn. The marker may be a bacteria, a chaperone mol., or a bacteria produced. The method may include a first assay and a second assay over a period for example of at least about 12 h. The concn. of HSP can be measured using an immunoassay. The assay may use a nucleotide mol. encoding HSP. Also provided herein is a method of treating or preventing **osteoporosis** caused by a bone disease which includes administering to a mammalian subject a therapeutically effective amt. of a formulation which is either an HSP antigenic

formulation or a bacterial antigenic formulation. The **osteoporosis** can be caused by a bone disease induced by bone infectious agents such as viruses, bacteria, fungi, protozoa and parasites. The HSP can be further complexed with an antigenic material or formulated in combination with an adjuvant. The antigenic material can be a peptide or a protein having an antigenic determinant of a virus, bacteria, fungi, protozoa or parasite that induces a bone disease. The antigenic material includes an antigenic determinant of a virus. Further, the methods disclosed herein can be practiced using a kit formed according to the methods disclosed herein.

L29 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:640075 HCAPLUS
TITLE: Orally active p38 kinase inhibitors from the urea class
AUTHOR(S): Dumas, Jacques; Hatoum-Mokdad, Holia; Sibley, Robert N.; Smith, Roger A.; Scott, William J.; Lee, Wendy; Wood, Jill; Wolanin, Donald; Cooley, Jeffrey; Bankston, Donald; Redman, Aniko; Schoenleber, Robert; Caringal, Yolanda; Housley, Timothy J.; Wilhelm, Scott M.; Pirro, John; Chien, Du-Shieng; Ranges, Gerald E.; Shrikhande, Alka; Muszi, Andrew; Bortolon, Elizabeth; Wakefield, Jean; Gianpaolo-Ostravage, Cynthia; Chau, Thuy
CORPORATE SOURCE: Bayer Research Center, Bayer Corporation, Pharmaceutical Division, West Haven, CT, 06516, USA
SOURCE: Abstracts of Papers, 222nd ACS National Meeting, Chicago, IL, United States, August 26-30, 2001 (2001), MEDI-256. American Chemical Society: Washington, D. C.
CODEN: 69BUZP
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English
AB The MAP kinase p38 is involved in IL-1 and TNF signaling pathways, and provides a novel approach to the treatment of **osteoporosis** and inflammatory disorders. For example, the pyridyl imidazole SB 203580 shows potent activity in models of **endotoxin** shock and bone resorption. **Screening** of a Bayer combinatorial chem. library afforded a series of pyrazolyl ureas as reversible inhibitors of p38. Optimization using parallel synthesis techniques resulted in the identification of N- (3-tert-butyl-1-methyl-5-pyrazolyl) -N'- (4-(4-pyridinylmethyl) phenyl)urea. This analog is a potent and selective inhibitor of p38 kinase biochem. and cellular assays. It is orally available in mice, and shows significant in vivo activity in two acute models of cytokine release (TNF induced IL-6 and LPS-induced TNF) and a chronic model of arthritis (20-day murine collagen induced arthritis).

L29 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:265451 HCAPLUS
DOCUMENT NUMBER: 134:290383
TITLE: Novel human G-protein coupled receptor for drug **screening**
INVENTOR(S): Deleersnijder, Willy; Berger, Claudia; Loeken, Christiane; Nys, Guy; Venema, Jacob
PATENT ASSIGNEE(S): Solvay Pharmaceuticals B.V., Neth.
SOURCE: PCT Int. Appl., 102 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025269	A2	20010412	WO 2000-EP9584	20000925
WO 2001025269	A3	20011011		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1218412	A2	20020703	EP 2000-984940	20000925

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

JP 2003521894	T2	20030722	JP 2001-528209	20000925

PRIORITY APPLN. INFO.:

EP 1999-203140	A	19990924
NL 1999-1013140	A	19990924
EP 2000-202683	A	20000728
US 2000-222047P	P	20000731
WO 2000-EP9584	W	20000925

AB The present invention relates to novel identified polynucleotides, polypeptides encoded by them and to the use of such polynucleotides and polypeptides, and to their prodn. More particularly, the polynucleotides and polypeptides of the present invention relate to the G-protein coupled receptor family, referred to as IGS4-family. The invention also relates to inhibiting or activating the action of such polynucleotides and polypeptides, to a vector contg. said polynucleotides, a host cell contg. such vector and transgenic animals where the IGS4-gene is either overexpressed, misexpressed, underexpressed or suppressed (knock-out animals). The invention further relates to a method for **screening** compds. capable to act as an agonist or an antagonist of said G-protein coupled receptor family IGS4 and the use of IGS4 polypeptides and polynucleotides and agonists or antagonists to the IGS4 receptor family in the treatment of PNS, psychiatric and CNS disorders, including schizophrenia, episodic paroxysmal anxiety EPA disorders such as obsessive compulsive disorder OCD, post traumatic stress disorder PTSD, phobia and panic, major depressive disorder, bipolar disorder, Parkinson's disease, general anxiety disorder, autism, delirium, multiple sclerosis, Alzheimer disease/dementia and other neurodegenerative diseases, severe mental retardation, dyskinesias, Huntington's disease, Tourett's syndrome, tics, tremor, dystonia, spasms, anorexia, bulimia, stroke, addiction/dependency/craving, sleep disorder, epilepsy, migraine; attention deficit/hyperactivity disorder (ADHD); cardiovascular diseases, including heart failure, angina pectoris, arrhythmias, myocardial infarction, cardiac hypertrophy, and hypotension. Also disclosed are hypertension - e.g., essential hypertension, renal hypertension, or pulmonary hypertension, thrombosis, arteriosclerosis, cerebral vasospasm, subarachnoid hemorrhage, cerebral ischemia, cerebral infarction, peripheral vascular disease, Raynaud's disease, kidney disease - e.g. renal failure; dyslipidemias; obesity; emesis; gastrointestinal disorders, including irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), gastroesophagal reflux disease (GERD), motility disorders and conditions of delayed gastric emptying, such as post operative or diabetic gastroparesis, and diabetes, ulcers, e.g., gastric ulcer; diarrhea; other diseases including **osteoporosis**; inflammations; infections such as bacterial, fungal, protozoan and viral infections, particularly

infections caused by HIV-1 or HIV-2; pain; cancers; chemotherapy induced injury; tumor invasion; immune disorders; urinary retention; asthma; allergies; arthritis; benign prostatic hypertrophy; **endotoxin** shock; sepsis; complication of diabetes mellitus; and gynaecol. disorders, among others and diagnostic assays for such conditions. Preferred uses of the invention relate to disorders of the nervous system, including the central nervous system CNS and the peripheral nervous system PNS, disorders of the gastrointestinal system and/or of the cardiovascular system and/or of skeletal muscle and/or of the thyroid, and/or also to lung diseases, immunol. diseases and disorders of the genitourinary system. The invention also relates to the identification of the cognate ligand of the IGS4 polypeptides of the invention. High affinity binding to said IGS4 polypeptides is found for the neuropeptides known as neuromedin U.

L29 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1996:756546 HCAPLUS
DOCUMENT NUMBER: 126:17804
TITLE: Human antibodies derived from immunized xenomice
INVENTOR(S): Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue; Brenner, Daniel G.; Capon, Daniel J.
PATENT ASSIGNEE(S): Cell Genesys, Inc., USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9634096	A1	19961031	WO 1995-US5500	19950428
W: AU, CA, FI, HU, JP, KR, NO, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2219486	AA	19961031	CA 1995-2219486	19950428
AU 9524668	A1	19961118	AU 1995-24668	19950428
EP 823941	A1	19980218	EP 1995-918935	19950428
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
JP 11505107	T2	19990518	JP 1995-532463	19950428

PRIORITY APPLN. INFO.: WO 1995-US5500 19950428

AB Antibodies with fully human variable regions against a specific antigen can be prepd. by administering the antigen to a transgenic animal which has been modified to produce such antibodies in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either antibodies per se or analogs thereof.

=> d ibib abs l31 1-28

=> d que stat l29
L10 13791 SEA FILE=HCAPLUS ABB=ON ?OSTEOPOROS? OR ?BONE?(W)?LOSS?
L12 1110 SEA FILE=HCAPLUS ABB=ON L10 AND (?SCREENING? OR ?DIAGNOSIS?)
L13 557 SEA FILE=HCAPLUS ABB=ON L10 AND ?SCREENING?
L15 2 SEA FILE=HCAPLUS ABB=ON L13 AND ?ENDOTOXIN?
L19 3 SEA FILE=HCAPLUS ABB=ON L12 AND ?ENDOTOXIN?
L20 1 SEA FILE=HCAPLUS ABB=ON L12 AND ?GAPSTATIN?
L21 1 SEA FILE=HCAPLUS ABB=ON L12 AND ?DERMONECROT?
L24 4 SEA FILE=HCAPLUS ABB=ON L15 OR L19 OR L20 OR L21
L25 1 SEA FILE=HCAPLUS ABB=ON L12 AND (S OR ?STAPHYLOCOCCUS?)(W)?AUR EUS?
L26 1 SEA FILE=HCAPLUS ABB=ON L12 AND ?BRONCHISEPTICA?
L27 1 SEA FILE=HCAPLUS ABB=ON L12 AND ?FUSOBACTERIUM?
L28 1 SEA FILE=HCAPLUS ABB=ON L25 OR L26 OR L27
L29 4 SEA FILE=HCAPLUS ABB=ON L24 OR L28

L31 ANSWER 1 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 2003184517 EMBASE
TITLE: Japanese guidelines for **diagnosis** and treatment of junctional and dystrophic epidermolysis bullosa.
AUTHOR: Tamai K.; Hashimoto I.; Hanada K.; Ikeda S.; Imamura S.; Ogawa H.
CORPORATE SOURCE: K. Tamai, Hirosaki Univ. School of Medicine, Hirosaki, Japan. katsuto@cc.hirosaki-u.ac.jp
SOURCE: Archives of Dermatological Research, Supplement, (2003) 295/1 (S24-S28).
Refs: 20
ISSN: 0944-1948 CODEN: ADRSFV
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index
LANGUAGE: English

L31 ANSWER 2 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-130873 [17] WPIDS
DOC. NO. CPI: C2002-040251
TITLE: Novel human G-protein coupled receptor IGPcR11 polypeptide and polynucleotide, useful for diagnosing, preventing, ameliorating and treating psychiatric and central nervous system diseases.
DERWENT CLASS: B04 D16
INVENTOR(S): NEHLS, M C; TROMMLER, P; WATTLER, F; WATTLER, S
PATENT ASSIGNEE(S): (INGE-N) INGENIUM PHARM AG
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002000689	A2	20020103	(200217)*	EN	62
AU 2001081936	A	20020108	(200235)		

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002000689 A2		WO 2001-EP7544	20010702
AU 2001081936 A		AU 2001-81936	20010702

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001081936 A	Based on	WO 200200689

PRIORITY APPLN. INFO: US 2000-215880P 20000630
AN 2002-130873 [17] WPIDS
AB WO 200200689 A UPAB: 20020313
NOVELTY - Human IGPcR11 protein (a G-protein coupled receptor) (I) comprising a 281 residue amino acid sequence (S1), fully defined in the specification, or its unique fragment of sequence greater than ten amino acids in length including polypeptides, peptides, isolated domains and fusion proteins, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated nucleic acid molecule (II) comprising an 846 nucleotide sequence (S2), fully defined in the specification, a nucleotide sequence which encodes (I), or an allelic variant of a nucleotide sequence encoding S1, where the allelic variant contains at least 80% nucleic acid homology and hybridizes to the complement of S2 under highly stringent conditions equivalent to hybridization in 42 deg. C in a hybridization solution comprising 50 % formamide, 1 % SDS (sodium dodecyl sulfate), 1M NaCl, 10 % Dextran sulfate and washing twice for 30 minutes in a wash solution comprising 0.1 multiply SSC (saline sodium chloride) and 1 % SDS;

(2) a vector (III) comprising (II);

(3) a host cell (IV) genetically engineered to contain (II) or (III);

(4) antibodies (Abs) to (I);

(5) agonists and antagonists (V) of (I) that compete selectively with native natural IGPcR11 ligand and which modulate IGPcR11 gene expression or gene product activity, including small molecules of molecular mass less than 6 kDa, molecules of intermediate size having molecular mass between 5 kDa-15 kDa and large molecules of molecular mass greater than 12 kDa, where the large molecules include mutant natural IGPcR11 ligand proteins that compete with native natural IGPcR11 ligand and which modulate IGPcR11 gene expression or gene product activity;

(6) antisense and ribozyme molecules (VI) that can be used to inhibit IGPcR11 gene expression or expression constructs used to enhance IGPcR11 gene expression;

(7) identifying (V) or (VI) which modulate the activity of IGPcR11 or IGPcR11 gene expression;

(8) embryonic stem cells containing a disrupted endogenous IGPcR11 gene;

(9) non-human knockout animals (VIIa) that do not express IGPcR11, where the endogenous animal ortholog of the IGPcR11 gene is functionally disrupted;

(10) mutated non-human animals (VIIb) that express a non-functional or partially functional form of IGPcR11;

(11) non-human transgenic animal (VIIc) model expressing the human IGPcR11 cDNA sequence comprising S2 or (II);

(12) progeny (VIId) of (VIIa)-(VIIc) including both heterozygous and homozygous offspring; and

(13) identifying compounds suitable for modulating the activity of (I), for the treatment of disease characterized by aberrant expression or activity of IGPcR11.

ACTIVITY - Neuroleptic; AntiParkinsonian; Tranquilizer; Neuroprotective; Nootropic; Anticonvulsant; Cerebroprotective; Antimigraine; Cardiant; Antianginal; Antiarrhythmic; Hypotensive; Antithrombotic; Antiarteriosclerotic; Vasotropic; Antilipemic; Anorectic; Antiinflammatory; Antidiarrheic; Antidiabetic; Antiulcer; Osteopathic; Antibacterial; Fungicide; Protozoacide; Virucide; Anti-HIV (human immunodeficiency virus); Analgesic; Cytostatic; Immunosuppressive; Antiasthmatic; Antiallergic; Antiarthritic; Antimanic; Antidepressant; Relaxant; Metabolic; Hemostatic.

MECHANISM OF ACTION - Gene therapy; modulator of IGPcR11 expression or activity (claimed).

No biological data is given.

USE - The transgenic non-human animals are useful for the dissection of the molecular mechanisms of IGPCR11 pathway for the identification and cloning of genes able to modify, reduce or inhibit the phenotype associated with IGPcR11 activity or deficiency. The animals are also useful for the identification of gene and protein diagnostic markers for diseases, for the identification and testing of compounds useful in the prevention, amelioration or treatment of disease associated with IGPcR11 activity or deficiency e.g. visual dysfunction associated with signal processing in the occipital lobe of the brain. The compounds that bind to modulate IGPcR11 gene or protein are useful for preventing, ameliorating or treating diseases characterized by aberrant IGPcR11 expression or activity. (III) and (IV) are useful in gene therapy. (All claimed). (II) is useful chromosomal mapping. IGPcR11 molecules are useful in **diagnosis**, prevention and amelioration of psychiatric and central nervous system (CNS) diseases including schizophrenia, obsessive compulsive disorder (COD), post-traumatic stress disorders (PTSD), phobia and panic, major depressive disorder, bipolar disorders, general anxiety disorder, autism, delirium, multiple sclerosis, Alzheimer's disease/dementia and other neurodegenerative diseases, severe mental retardation, visual diseases, movement diseases, Tourette's syndrome, Parkinson's disease, Huntington's disease, dyskinesias, dystonia, pain, spasms, anorexia, bulimia, stroke, addition/dependency/craving, sleep disorders, epilepsy, migraine, attention deficit/hyperactivity (ADHD), cardiovascular diseases including angina pectoris, heart failure, arrhythmias, myocardial infarction, cardiac hypertrophy, hypertension, thrombosis, arteriosclerosis, cerebral vasospasm, subarachnoid hemorrhage, cerebral ischemia, thrombosis, peripheral vascular disease, Raynaud's disease, kidney disease e.g. renal failure, dyslipidemias, obesity, emesis, gastrointestinal disorders including irritable bowel syndrome (IBS), inflammatory bowel syndrome (IBD), diarrhea, gastro-esophageal reflux disease (GERD), motility disorders and conditions of delayed gastric emptying, such as post operative or diabetic gastroparesis and diabetic ulcers, other diseases including **osteoporosis**, inflammations, infections such as bacterial, fungal, protozoan and viral infections (e.g. human immunodeficiency virus (HIV)-1 or HIV-2), pain, cancers, chemotherapy induced injury, tumor invasion, immune disorders, autoimmune diseases, urinary retention, asthma, allergies, arthritis, benign prostatic hypertrophy, **endotoxin** shock, sepsis, complication of diabetes mellitus and gynecological disorders.
Dwg.0/7

L31 ANSWER 3 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-130822 [17] WPIDS
DOC. NO. CPI: C2002-040202
TITLE: New G protein-coupled receptor (IGPc20) gene useful for

drug **screening**, and diagnosing or treating diseases, e.g. cancer, reproductive disorders and infertility related to epididymal dysfunction or inflammations.

DERWENT CLASS: B04 D16
INVENTOR(S): NEHLS, M C; TROMMLER, P; WATTLER, F; WATTLER, S
PATENT ASSIGNEE(S): (INGE-N) INGENIUM PHARM AG
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002000001	A2	20020103	(200217)*	EN	75

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001087574 A 20020108 (200235)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002000001	A2	WO 2001-EP7533	20010702
AU 2001087574	A	AU 2001-87574	20010702

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001087574 A	Based on	WO 200200001

PRIORITY APPLN. INFO: US 2000-215777P 20000630
AN 2002-130822 [17] WPIDS
AB WO 200200001 A UPAB: 20020313

NOVELTY - An isolated human G protein-coupled receptor (IGPcR20) nucleic acid comprises:

(a) a sequence:

(i) of 1011 (I) or 987 base pairs (bp) (II), given in the specification; or

(ii) encoding a polypeptide of 336 (III) or 328 amino acids (IV), given in the specification, or a fragment of (III) or (IV); or

(b) an allelic variant of the nucleotide sequence, which encodes a polypeptide comprising (III) or (IV), is new.

DETAILED DESCRIPTION - A new isolated nucleic acid molecule comprises:

(a) a nucleotide sequence:

(i) having 1011 base pairs (I) or 987 bp (II), given in the specification; or

(ii) encoding a polypeptide having 336 amino acids (III) or 328 amino acids (IV), given in the specification, or any unique fragment of (III) or (IV) that is greater than ten amino acids in length; or

(b) an allelic variant of the nucleotide sequence, which encodes a polypeptide comprising (III) or (IV).

The allelic variant contains 80 % nucleic acid homology and hybridizes to the complement of (I) under highly stringent conditions equivalent to hybridization in 42 deg. C in a hybridization solution comprising 50 % formamide, 1 % sodium dodecyl sulfate (SDS), 1M NaCl, 10%

Dextran sulfate, and washing twice for 30 minutes in a wash solution comprising 0.1 multiply saline sodium citrate (SSC) and 1 % SDS.

INDEPENDENT CLAIMS are also included for the following:

(1) a vector comprising the isolated nucleic acid molecule;

(2) a host cell genetically engineered to contain the nucleic acid molecule or the vector;

(3) the human G protein-coupled receptor (IGPcR) protein, IGPcR20 of (III), the mouse IGPcR20 protein of (IV), or any of their unique fragments with a sequence having greater than ten amino acids in length, including but not limited to polypeptides, peptides, isolated domains or fusion proteins;

(4) antibodies specifically targeting the IGPcR20 proteins, and/or polypeptides, peptides, isolated domains, and the IGPcR20 component of fusion proteins of the IGPcR20 proteins;

(5) agonists and antagonists of IGPcR20 protein that compete selectively with native natural IGPcR20 ligand and which modulate IGPcR20 gene expression or gene product activity, including:

(a) small molecules of molecular mass less than 6 kDa;

(b) molecules of intermediate size, having a molecular mass between 5 - 15 kDa; and

(c) large molecules of molecular mass greater than 12 kDa, the latter including mutant natural IGPcR20 ligand proteins that compete with native natural IGPcR20 ligand and which modulate IGPcR20 gene expression or gene product activity;

(6) antisense and ribozyme molecules that can be used to inhibit IGPcR20 gene expression or expression constructs used to enhance IGPcR20 gene expression;

(7) methods of identifying compounds of (5) or (6), which modulate the activity of IGPcR20 or IGPcR20 gene expression;

(8) embryonic stem cells containing the disrupted endogenous IGPcR20 gene;

(9) non-human knock-out animals that do not express IGPcR20, where the endogenous animal ortholog of the IGPcR20 gene is functionally disrupted;

(10) mutated non-human animals that express a non-functional or partially functional form of IGPcR20;

(11) a non-human transgenic animal model expressing the human IGPcR20 cDNA sequence, or (I) or (II);

(12) progeny of the non-human animals, including both heterozygous and homozygous offspring;

(13) identifying compounds for modulating the activity of the protein for treating diseases characterized by aberrant expression or activity of IGPcR20; and

(14) a method or a gene therapy method of preventing, ameliorating or treating diseases characterized by aberrant expression or activity of IGPcR20 by administrating compounds that specifically bind to the IGPcR20 gene or protein and /or which modulate IGPcR20 expression or activity, or by administrating vectors and/or host cells containing nucleotide sequences (the new nucleic acid, (1) and (2)) that modulate IGPcR20 expression or activity.

ACTIVITY - Vasotropic; cardiovascular; cytostatic; neuroprotective; cerebroprotective; neuroleptic; nephrotropic; gastrointestinal; antiinflammatory; immunosuppressive; antiallergic; gynecological; antiinfertility; immunostimulant; tranquilizer; antidepressant; antiParkinsonian; nootropic; anticonvulsant; antimigraine; antianginal; anticoagulant; thrombolytic; vasotropic; osteopathic; antibacterial; antidiabetic. No biological data is given.

MECHANISM OF ACTION - Gene therapy; human G protein-coupled receptor (IGPcR20) agonist/antagonist.

USE - The IGPcR20 gene or protein encoded by it is useful for drug

screening, and diagnosing or treating diseases and disorders, particularly cancer, reproductive disorders and infertility related to epididymal dysfunction, pain, or metabolic and inflammatory disorders. In particular, the IGPcR20 gene or protein is useful for treating or diagnosing psychiatric and central nervous system (CNS) disorders (e.g. schizophrenia, episodic paroxysmal anxiety (EPA), phobia or panic, major depressive disorder, Parkinson's disease, Alzheimer's disease/dementia and neurodegenerative diseases, severe mental retardation, dyskinesias, Huntington's disease, Gille de la Tourette's syndrome, sleep disorders, epilepsy, migraine, or attention deficit/hyperactivity disorder), cardiovascular diseases (e.g. angina pectoris, cerebral vasospasm, thrombosis or Raynaud's disease), kidney disease, gastrointestinal disorders, osteoporosis, inflammation, infection, immune disorders, autoimmune diseases, allergies, endotoxin shock, sepsis, complication of diabetes mellitus, or gynecological and reproductive disorders and male infertility. A non-human animal is useful for the dissection of the molecular mechanisms of the IGPcR20 pathway, and for the identification and cloning of genes able to modify, reduce or inhibit the phenotype associated with IGPcR20 activity or deficiency. The animal model is useful for the identification of gene and protein diagnostic markers for diseases, for the identification and testing of compounds useful in the prevention, amelioration or treatment of diseases associated with IGPcR20 activity or deficiency. The disease comprises diseases associated with signal processing in male reproductive tissues, particularly testis or epididymis (all claimed).
Dwg.0/10

L31 ANSWER 4 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-215262 [27] WPIDS
DOC. NO. CPI: C2002-065748
TITLE: An isolated polypeptide with phosphohydrolase activity, designated CD39L2, useful to identify other proteins with which binding occurs or identify inhibitors and for treatment of, e.g., Alzheimer's, multiple sclerosis and **osteoporosis**.
DERWENT CLASS: B04 D16
INVENTOR(S): CHADWICK, B P; FRISCHAUF, A
PATENT ASSIGNEE(S): (HYSE-N) HYSEQ INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6350447	B1	20020226	(200227)*		101

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 6350447	B1	US 1999-240639	19990129

PRIORITY APPLN. INFO: US 1999-240639 19990129
AN 2002-215262 [27] WPIDS
AB US 6350447 B UPAB: 20020429
NOVELTY - An isolated polypeptide (I) with phosphohydrolase activity comprising an amino acid sequence that has at least about 90% sequence identity to the fully defined 456 amino acid sequence (S1) as given in the specification, is new.
ACTIVITY - Immunomodulatory; virucide; antibacterial; antifungal;

neuroprotective; dermatological; immunosuppressive; antiinflammatory; antirheumatic; antiarthritic; antithyroid; antidiabetic; antiasthmatic; respiratory; vulnerary; antiulcer; periodontal; osteopathic; nootropic; anticonvulsant; nephrotropic; gastrointestinal; vasotropic.

No supporting data given.

MECHANISM OF ACTION - None given in the source material.

USE - (I) is useful in an assay to determine biological activity, including in a panel of multiple proteins for high-throughput **screening**; to raise antibodies or to elicit another immune response; as a reagent in assays designed to quantitatively determine levels of the protein (or its receptor) in biological fluids; as markers for tissues in which the corresponding protein is preferentially expressed and to isolate correlative receptors or ligands. Where the protein binds or potentially binds to another protein (such as, for example, in a receptor-ligand interaction), the protein can be used to identify the other protein with which binding occurs or to identify inhibitors of the binding interaction.

In addition, (I) may be useful in the treatment of various immune deficiencies and disorders. More specifically, infectious diseases causes by viral, bacterial, fungal or other infection may be treatable with (I). Autoimmune disorders which may be treated using (I), for example, multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune thyroiditis and insulin dependent diabetes mellitus. (I) may also be useful in the treatment of allergic reactions and conditions, such as asthma or other respiratory problems. (I) may have utility in compositions used for bone, cartilage, tendon, ligament and/or nerve tissue growth or regeneration, as well as for wound healing and tissue repair and replacement, and in the treatment of burns, incisions and ulcers.

Moreover, (I) may also be used in the treatment of periodontal disease, and in other tooth repair processes and in the treatment of **osteoporosis** or osteoarthritis.

(I) may also be useful for proliferation of neural cells and for regeneration of nerve and brain tissue, i.e. for the treatment of central and peripheral nervous system diseases and neuropathies, as well as mechanical and traumatic disorders, which involve degeneration, death or trauma to neural cells or nerve tissue. More specifically, (I) may be used in the treatment of diseases of the peripheral nervous system, such as peripheral nerve injuries, peripheral neuropathy and localized neuropathies, and central nervous system diseases, such as Alzheimer's, Parkinson's disease, Huntington's disease and amyotrophic lateral sclerosis.

(I) may also be useful to promote better or faster closure of non-healing wounds, including pressure ulcers, ulcers associated with vascular insufficiency and surgical and traumatic wounds.

Furthermore, (I) may also exhibit anti-inflammatory activity and may be used to treat inflammatory conditions including chronic or acute conditions), including ischemia-reperfusion injury, **endotoxin** lethality, arthritis, nephritis, cytokine or chemokine-induced lung injury, inflammatory bowel disease or Crohn's disease.

Dwg.0/9

L31 ANSWER 5 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN

ACCESSION NUMBER: 2003083068 EMBASE

TITLE: Aortic stent-graft infection due to a presumed aortoenteric fistula.

AUTHOR: Kar B.; Dougherty K.; Reul G.J.; Krajcer Z.

CORPORATE SOURCE: Dr. Z. Krajcer, 6624 Fannin, Houston, TX 77030, United States. ZvonkoMD@aol.com

SOURCE: Journal of Endovascular Therapy, (2002) 9/6 (901-906). Refs: 15

ISSN: 1526-6028 CODEN: JENTFI
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
009 Surgery
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Purpose: To report a case of late stent-graft infection with aortoenteric fistula. Case Report: A 76-year-old Colombian man received an AneuRx stent-graft for a 5.5-cm infrarenal AAA. The aneurysm sac progressively shrank until 22 months postoperatively, when an increase in diameter was noted on magnetic resonance imaging without evidence of endoleak or air in the sac. Two months prior, the patient had developed fever and an elevated white blood cell count; he underwent a 6-week course of intravenous antibiotics. Shortly thereafter, the fever recurred, along with progressive weight loss, which prompted admission. The computed tomographic scan showed no evidence of endoleak, but gas collection was seen anteriorly in the sac; aspirated material was positive for a variety of organisms. At surgery 23 months after stent-graft implantation, pronounced inflammatory reaction and scarring were seen around the graft in conjunction with evidence of a healed duodenal perforation, suggestive of an aortoenteric fistula. The excised stent-graft was intact; no deterioration was seen. The patient had a protracted recovery but has been afebrile and asymptomatic > 1 year after stent-graft explantation Conclusions: Close surveillance after endovascular AAA repair is essential to detect late leaks, secondary migration, endotension, structural failure, and infection with or without aortoenteric fistula.

L31 ANSWER 6 OF 28 MEDLINE on STN
ACCESSION NUMBER: 2002285794 MEDLINE
DOCUMENT NUMBER: 22022823 PubMed ID: 12027261
TITLE: Treatment of periodontal disease in a patient with Ehlers-Danlos syndrome. A case report and literature review.
AUTHOR: Perez Luis A; Al-Shammari Khalaf F; Giannobile William V; Wang Hom-Lay
CORPORATE SOURCE: Department of Periodontics/Prevention/Geriatrics, University of Michigan School of Dentistry, Ann Arbor, USA.
SOURCE: JOURNAL OF PERIODONTOLOGY, (2002 May) 73 (5) 564-70. Ref: 48
Journal code: 8000345. ISSN: 0022-3492.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020528
Last Updated on STN: 20020810
Entered Medline: 20020809
AB BACKGROUND: Ehlers-Danlos syndrome (EDS) designates a heterogeneous group of connective tissue disorders characterized by skin elasticity, tissue fragility, and chronic joint pain. Dental findings have been reported with some types of EDS. This case report describes the periodontal findings in a patient with a previously undiagnosed EDS type VIII. METHODS: Diagnostic aids utilized included microbial testing, histological

examination, gingival crevicular fluid (GCF) analysis for the levels of C-telopeptide pyridinoline cross-links (ICTP), and genetic counseling. Periodontal treatment consisted of mechanical debridement and adjunctive antibiotic therapy. RESULTS: Genetic counseling and clinical presentation confirmed the **diagnosis** of EDS type VIII. Periodontal treatment led to marked clinical improvements and GCF levels of the bone resorptive marker ICTP were significantly reduced. The patient and her siblings are currently pursuing appropriate medical care and genetic counseling. CONCLUSION: Periodontal involvement may lead to the **diagnosis** of an underlying systemic condition. Identification of suspected etiological factors of periodontal disease may prove critical for the general well-being of some patients.

L31 ANSWER 7 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 2003038609 EMBASE
TITLE: Antithrombotic agents in the management of sepsis.
AUTHOR: Iqbal O.; Tobu M.; Hoppensteadt D.; Aziz S.; Messmore H.; Fareed J.
CORPORATE SOURCE: Dr. O. Iqbal, Loyola University, Medical Center, Maywood, IL 60153, United States
SOURCE: Turkish Journal of Haematology, (2002) 19/3 (349-389). Refs: 345
ISSN: 1300-7777 CODEN: TJHSFS
COUNTRY: Turkey
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
030 Pharmacology
037 Drug Literature Index
039 Pharmacy
029 Clinical Biochemistry
025 Hematology
018 Cardiovascular Diseases and Cardiovascular Surgery
038 Adverse Reactions Titles
036 Health Policy, Economics and Management
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Sepsis, a systemic inflammatory syndrome, is a response to infection and when associated with multiple organ dysfunction is termed, severe sepsis. It remains a leading cause of mortality in the critically ill. The response to the invading bacteria may be considered as a balance between proinflammatory and antiinflammatory reaction. While an inadequate proinflammatory reaction and a strong antiinflammatory response could lead to overwhelming infection and death of the patient, a strong and uncontrolled proinflammatory response, manifested by the release of proinflammatory mediators may lead to microvascular thrombosis and multiple organ failure. **Endotoxin** triggers sepsis by releasing various mediators including tumor necrosis factor-alpha and interleukin-1(IL-1). These cytokines activate the complement and coagulation systems, release adhesion molecules, prostaglandins, leukotrienes, reactive oxygen species and nitric oxide (NO). Other mediators involved in the sepsis syndrome include IL-1, IL-6 and IL-8; arachidonic acid metabolites; platelet activating factor (PAF); histamine; bradykinin; angiotensin; complement components and vasoactive intestinal peptide. These proinflammatory responses are counteracted by IL-10. Most of the trials targeting the different mediators of proinflammatory response have failed due a lack of correct definition of sepsis. Understanding the exact pathophysiology of the disease will enable better treatment options. Targeting the coagulation system with various anticoagulant agents including antithrombin, activated protein C (APC), tissue factor pathway inhibitor (TFPI) is a rational approach. Many

clinical trials have been conducted to evaluate these agents in severe sepsis. While trials on antithrombin and TFPI were not so successful, the double-blind, placebo-controlled, phase III trial of recombinant human activated protein C worldwide evaluation in severe sepsis (PROWESS) was successful, significantly decreasing mortality when compared to the placebo group. Better understanding of the pathophysiologic mechanism of severe sepsis will provide better treatment options. Combination antithrombotic therapy may provide a multipronged approach for the treatment of severe sepsis.

L31 ANSWER 8 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-122283 [16] WPIDS
DOC. NO. CPI: C2002-037518
TITLE: Novel purified human eosinophil serine protease 1-like enzyme, useful for identifying modulators of enzyme activity for treating Paget's disease, **osteoporosis**, airway allergy, asthma.
DERWENT CLASS: B04 D16
INVENTOR(S): XIAO, Y
PATENT ASSIGNEE(S): (FARB) BAYER AG; (XIAO-I) XIAO Y
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001098503	A2	20011227	(200216)*	EN	131
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001083872	A	20020102	(200230)		
US 2002146407	A1	20021010	(200269)		
EP 1297158	A2	20030402	(200325)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001098503	A2		WO 2001-EP6936	20010620
AU 2001083872	A		AU 2001-83872	20010620
US 2002146407	A1	Provisional	US 2000-212844P	20000621
		Provisional	US 2000-244171P	20001031
		Provisional	US 2001-279766P	20010330
			US 2001-885441	20010621
EP 1297158	A2		EP 2001-962751	20010620
			WO 2001-EP6936	20010620

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001083872	A	Based on	WO 200198503
EP 1297158	A2	Based on	WO 200198503

PRIORITY APPLN. INFO: US 2001-279766P 20010330; US 2000-212844P 20000621; US 2000-244171P 20001031

AN 2002-122283 [16] WPIDS
AB WO 200198503 A UPAB: 20020308
NOVELTY - A purified human eosinophil serine protease (esp) 1-like enzyme (I) having a a 339, 279, 334, 305 or 259 residue amino acid sequence, fully defined in the specification, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (II):

(a) encoding (I) which comprises an amino acid sequence of PS or an amino acid sequence which is at least 52 % identical to PS;

(b) comprising a 1018 (S1) or 837 (S8) nucleotide sequence, fully defined in the specification;

(c) which hybridizes under stringent conditions to an above mentioned polynucleotide;

(d) having a sequence which deviates from the above mentioned sequences due to degeneracy of genetic code; or

(e) which represents a fragment, derivative or allelic variant of the above mentioned polynucleotide sequences;

(2) an expression vector (III) containing (II);

(3) a host cell (IV) containing (III);

(4) a substantially purified esp 1-like polypeptide (P1) encoded by (II);

(5) preparation of (I), comprising culturing (IV) under expression conditions, and recovering the polypeptide;

(6) a reagent (V) that modulates the activity of (I) or (II), which is identified by **screening** methods involving (P1) or (I), or (II);

(7) a diagnostic kit for detecting a coding sequence for a polypeptide comprising an amino acid sequence of PS, comprising a polynucleotide having 11 contiguous nucleotides of (S1) or (S8) and instructions of carrying out the detection method;

(8) a kit for detecting a polypeptide comprising an amino acid sequence of PS, comprising an antibody which specifically binds to (I) and instructions for carrying out the method;

(9) a pharmaceutical composition (VI) comprising (III) or (V) which specifically binds to (I) having an amino acid sequence of PS, or to (II) and modulates their activity, identified by **screening** methods involving (P1) or (I), or (II); and a carrier;

(10) a fusion protein comprising (I);

(11) **screening** (M1) for agents which modulate an activity of a human esp 1-like enzyme comprising contacting a test compound with a product encoded by a polynucleotide which comprises the nucleotide sequence of (S1) or (S8) and detecting binding of the test compound of the product, where a test compound which binds to the product is identified as a potential agent for regulating the activity of the human esp 1-like enzyme;

(12) reducing (M2) activity of human esp 1-like enzyme comprising contacting a cell with a reagent which specifically binds to a product encoded by a polynucleotide comprising the nucleotide sequence of (S1) or (S8); and

(13) a pharmaceutical composition comprising a reagent which binds to a product of a polynucleotide comprising the nucleotide sequence of (S1) or (S8) and a carrier.

ACTIVITY - Antiinflammatory; antiasthmatic; antiallergic; osteopathic; cytostatic; dermatological.

Synthesis of an antisense esp 1-like enzyme oligonucleotide comprising at least 11 contiguous nucleotides from the complement of a 1018 nucleotide sequence, fully in the specification was performed on a Pharmacia Gene Assembler series synthesizer. Following assembly and deprotection, the oligonucleotide was twice ethanol-precipitated, dried

and suspended in phosphate-buffered saline (PBS) at the desired concentration. Purity of the oligonucleotides was tested by capillary gel electrophoresis and ion exchange high performance liquid chromatography (HPLC). The **endotoxin** level in the oligonucleotide preparation was determined using the Limulus Amebocyte assay. An aqueous composition containing the antisense oligonucleotide at a concentration of 0.1-100 micro m was administered directly to a patient having asthma by injection. The severity of the patient's asthma was decreased.

MECHANISM OF ACTION - esp 1-like enzyme activity modulator.

USE - (I) is useful for **screening** for agents which decrease the activity of an esp 1-like enzyme which involves contacting a test compound with (P1) or (II), and detecting binding of test compound to polypeptide or polynucleotide. The test compound which binds to the polypeptide or polynucleotide is identified as a potential therapeutic agent for decreasing the activity of esp 1-like enzyme. (I) is also useful for **screening** for agents which regulate the activity of an esp 1-like enzyme. The method involves contacting a test compound with (P1) or (I) and detecting the esp 1-like enzyme activity of the polypeptide. The test compound which increases or decreases the esp 1-like enzyme activity is identified as a potential therapeutic for increasing or decreasing the esp 1-like enzyme activity. The method optionally involves detecting binding of the test compound to the polypeptide and a test compound which binds to the polypeptide is identified as a potential agent for regulating activity of the human esp 1-like enzyme. The polypeptide is contacted with the test compound in a cell, in vitro or in a cell-free system. Either the polypeptide or the test compound comprises a detectable label and the test compound when binding to the polypeptide displaces the labeled ligand bound to the polypeptide. Also, the polypeptide or the test compound is bound to a solid support. (II) is useful for detecting a polynucleotide which encodes (I) in a biological sample which involves hybridizing a polynucleotide comprising 11 contiguous nucleotides of (S1) or (S8) to a nucleic acid material of a biological sample, thereby forming a hybridization complex and detecting a hybridization complex. Before hybridization the nucleic acid material of the biological sample is amplified. (V) is useful for detecting (P1) or (II) which involves contacting a biological sample with (V). (V) is also useful for reducing the activity of esp 1-like enzyme which involves contacting a cell with (V) which binds to (II) or (P1). (V) (an antibody is useful for detecting the polypeptide having an amino acid sequence of PS. (V) is also useful for treating a esp 1-like enzyme dysfunction related diseases condition such as asthma, chronic obstructive pulmonary disease, airway allergy or **osteoporosis**. (VI) is useful for modulating esp 1-like enzyme activity in a disease condition as mentioned above. (All claimed). (I) is useful in diagnostic assays for detecting diseases and abnormalities or susceptibility to diseases and abnormalities related to presence of mutations in the nucleic acid sequences which encode the enzyme. (V) is also useful for treating dermatitis, Paget's disease, and preventing degradation of bone implants particularly dental implants.

Dwg.0/29

L31 ANSWER 9 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN

ACCESSION NUMBER: 2001-343815 [36] WPIDS

DOC. NO. NON-CPI: N2001-248980

DOC. NO. CPI: C2001-106500

TITLE: New IGS5 polypeptides useful for treating infections, pain, cancer, diabetes, obesity, anorexia, bulimia, asthma, schizophrenia, hypertension, urinary retention and Parkinson's disease.

DERWENT CLASS: B04 D16 S03

INVENTOR(S): DELEERSNIJDER, W; WESKE, M; WIEGERS, R

PATENT ASSIGNEE(S): (SOLV) SOLVAY PHARM BV
COUNTRY COUNT: 92
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001036610	A1	20010525	(200136)*	EN	114
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001018582	A	20010530	(200152)		
EP 1234025	A1	20020828	(200264)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
JP 2003514525	W	20030422	(200336)		135
CN 1399678	A	20030226	(200337)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001036610	A1	WO 2000-EP11532	20001117
AU 2001018582	A	AU 2001-18582	20001117
EP 1234025	A1	EP 2000-981279	20001117
		WO 2000-EP11532	20001117
JP 2003514525	W	WO 2000-EP11532	20001117
		JP 2001-538489	20001117
CN 1399678	A	CN 2000-815837	20001117

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001018582	A	Based on	WO 200136610
EP 1234025	A1	Based on	WO 200136610
JP 2003514525	W	Based on	WO 200136610

PRIORITY APPLN. INFO: NL 2000-1015356 20000531; EP 1999-203862 19991119; NL 1999-1013616 19991119; EP 2000-201937 20000531

AN 2001-343815 [36] WPIDS

AB WO 200136610 A UPAB: 20021031

NOVELTY - An isolated IGS5 (not defined) polypeptide (I) comprising a sequence having at least 70% identity to a sequence (S1) comprising 691, 779 or 753 amino acids fully defined in the specification, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated fragment (F1) of (I) showing at least one of the characteristic activities of metalloproteases or functionally related enzymes;

(2) an isolated polynucleotide (II) comprising:

(a) a sequence encoding (I);

(b) a sequence having at least 70% identity to a sequence encoding (I);

(c) a sequence having at least 70% identity to the 2076, 2340 or 2262 base pair (bp) sequence (S2) given in the specification; or

(d) a sequence obtainable by **screening** an appropriate

library under stringent hybridization conditions with a labeled probe comprising S2, or its complements;

(3) an isolated polynucleotide fragment (F2) encoding F1;

(4) an expression system (III) comprising (II) capable of producing (I), when present in a compatible host cell;

(5) a host cell (IV) comprising (III), or a membrane (IVa) expressing (I);

(6) producing (I);

(7) an antibody (Ab) immunospecific for (I);

(8) **screening** to identify compounds which influence the activity of (I) or F1, by contacting (IV), (IVa), (I) or F1 with a candidate compound (CC) and assessing whether the compound results in a stimulation or inhibition of the activity of (I);

(9) **screening** (M1) to identify compounds which stimulate or inhibit the function or level of (I) or F1, comprising:

(a) measuring the influence of CC on the activity of (I), (IV), (IVa) or a fusion protein comprising (I), in the presence of a suitable substrate for (I);

(b) measuring the influence of CC on the activity of (I), (IV), (IVa) or fusion protein comprising (I) in the presence of a competitor of (I);

(c) testing whether CC results in a signal generated by activation or inhibition of (I), using detection systems appropriate to the activity of (I), (IV) or (IVa);

(d) mixing CC with a solution comprising (I) and a suitable substrate, to form a mixture, measuring activity of (I) in the mixture and comparing the activity of the mixture to a standard without CC; or

(e) detecting the effect of CC on the production of mRNA encoding (I) and (I) in cells, using e.g. enzyme linked immunosorbant assay (ELISA);

(10) a stimulant or inhibitor (V) of (I) or F1 identified by (M1);

(11) a compound (C) for use in therapy, selected from (V) identified by (M1), (II), or a nucleic acid molecule that modulates expression of (II); and

(12) diagnosing a disease or susceptibility to disease in a subject related to expression or activity of (I), comprising determining the presence or absence of a mutation in a nucleotide sequence encoding (I) in the genome of the subject, and/or analyzing for the presence or amount of expression of (I) in a sample derived from the subject.

ACTIVITY - Cytostatic; antidiabetic; anorectic; antiasthmatic; antiparkisonian; hypotensive; antianginal; osteopathic; cerebroprotective; antimigraine; antiulcer; antiallergic; neuroprotective; nootropic; antidiarrheic; neuroleptic; antibacterial; antiviral; antifungal; antiprotozoal; analgesic; antiemetic; cardiant; hypertensive; tranquilizer; anticonvulsant; thrombolytic; anticoagulant; antiarteriosclerotic; vasotropic; antiinflammatory; antiarthritic; nephrotroic; immunomodulatory.

MECHANISM OF ACTION - Gene therapy; vaccine.

No supporting biological data given.

USE - The **screening** method is useful for identifying compounds suitable for the treatment and/or prophylaxis of cardiovascular diseases. (V), (I) (II), a nucleic acid molecular that inhibits (II), a peptide that competes with IGS5 polypeptide, a polypeptide that degrades IGS5 and/or a nucleic acid molecule that enhances the expression of a nucleotide sequence encoding for a polypeptide that degrades IGS5, are useful in the treatment of a subject having altered activity of IGS5, where an increase or enhanced activity of IGS5 is linked with cardiovascular diseases (claimed).

(I) and (II) are useful for treating bacterial, fungal, protozoan and viral infections, particularly infections caused by HIV-1 or HIV-2, pain, cancer, diabetes, obesity, anorexia, bulimia, asthma, Parkinson's disease, acute heart failure, hypotension, hypertension, urinary retention,

osteoporosis, angina pectoris, myocardial infarction, stroke, ulcers, allergies, benign prostatic hypertrophy, migraine, psychotic and neurological disorders including anxiety, schizophrenia, delirium, dementia, obsessive compulsive disorder (OCD), post traumatic stress disorder (PTSD), phobia and panic, major depressive disorder, bipolar disorder, general anxiety disorder, autism, multiple sclerosis, Alzheimer's disease, and other neurodegenerative diseases, addiction/dependency/craving, sleep disorder, epilepsy, attention deficit/hyperactivity disorder (ADHD), cardiovascular diseases including arrhythmias, cardiac hypertrophy, renal hypertension, pulmonary hypertension, thrombosis; arteriosclerosis, cerebrovasospasm, subarachnoid hemorrhage, cerebral ischemia, cerebral infarction, peripheral vascular disease, Raynaud's disease, kidney diseases such as renal failure, dyslipidemia, emesis, gastrointestinal disorders including irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), gastroesophagal reflux disease (GERD), motility disorders and conditions of delayed gastric emptying, post-operative or diabetic gastroparesis, diarrhea, inflammations, chemotherapy induced injury, tumor invasion, immune disorders, arthritis, **endotoxin** shock, sepsis, complications of diabetes mellitus, and severe mental retardation and dyskinesias, such as Huntington's disease or Gilles de la Tourette's syndrome. (II) is useful as diagnostic reagents, for chromosome and tissue localization studies, and as valuable tools for tissue expression studies. (I) is useful in **screening** assays, to identify membrane bound or soluble receptors. (I) and (II) are also useful as vaccines.
Dwg.0/10

L31 ANSWER 10 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 2001380216 EMBASE
TITLE: Emergency management: The acute painful joint.
AUTHOR: Sturrock R.
CORPORATE SOURCE: Prof. Dr. R. Sturrock, Centre for Rheumatic Disease, University Department of Medicine, Royal Infirmary, 10 Alexandra Parade, Glasgow G31 2ER, United Kingdom
SOURCE: CPD Journal Internal Medicine, (2001) 2/3 (82-85).
Refs: 5
ISSN: 1466-2914 CODEN: CINMFG
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 031 Arthritis and Rheumatism
004 Microbiology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB There are few rheumatological emergencies but the acute painful joint requires urgent investigation and treatment. Ten million people will attend their general practitioner every year with pain and stiffness referrable to the musculoskeletal system. The majority of these have Osteoarthritis (OA) (5 million) with Rheumatoid arthritis (RA) accounting for 400,000 of the total. The precise number of individuals presenting with an acute painful joint is unknown.

L31 ANSWER 11 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:264667 BIOSIS
DOCUMENT NUMBER: PREV200200264667
TITLE: XVII Academic Meeting of German-Speaking University Teachers of Gynecology and Obstetrics, Salzburg, Austria, October 3-5, 2001.
AUTHOR(S): German-Speaking University Teachers of Gynecology and Obstetrics

SOURCE: Gynaekologisch-Geburtshilfliche Rundschau, (Oktober, 2001) Vol. 41, No. 2, pp. 71-149. http://www.karger.com/journals/ggr/ggr_jh.htm. print.
Meeting Info.: XVII Academic Meeting of German-Speaking University Teachers of Gynecology and Obstetrics Salzburg, Austria October 03-05, 2001
ISSN: 1018-8843.
DOCUMENT TYPE: Conference
LANGUAGE: German

AB This meeting contains 171 abstracts on various topics in obstetrics and gynecology, including 100 posters. The abstracts are all written in German. The abstracts describe human medicine and treatment of human gynecological diseases, but some abstracts have animal models. The main divisions of topics were feto-maternal medicine, gynecological oncology, breast cancer, endocrinology and reproductive medicine, and stem cells. Methods described include fetal MRI, ultrasound sonography, denaturing HPLC, laparoscopy, ELISA, FISH, immunohistochemistry, prenatal infection **screening**, and quantitative fluorescence PCR. Some of the diseases discussed include diabetes, chemotherapy resistance, pregnancy-induced hypertension, fetal **endotoxin** shock, vulvular cancer, **osteoporosis**, peritoneal endometriosis, HIV, HPV, placenta failure, syphilis, ectopic pregnancy, incontinence, fetal heart failure, and sudden infant death syndrome. Treatments discussed include tension-free vaginal tape therapy, ovarian tissue banking, hormone replacement therapy, caesarean section, labor induction, cancer surgery, and laparoscopic pelvic neurostimulation. Some of the therapy drugs discussed include Gemeprost, Survivin, Imiquimod, Epirubicin, Taxol, Paclitaxel, Herceptin, Adriamycin, Docetaxel, and Isosorbide-5 mononitrate.

L31 ANSWER 12 OF 28 MEDLINE on STN
ACCESSION NUMBER: 2000483448 MEDLINE
DOCUMENT NUMBER: 20329366 PubMed ID: 10872965
TITLE: Features of severe periodontal disease in a teenager with Chediak-Higashi syndrome.
AUTHOR: Delcourt-Debruyne E M; Boutigny H R; Hildebrand H F
CORPORATE SOURCE: Faculte d'Odontologie, Departement de Parodontologie, Universite Lille, France.. edelcourt@univ.lille2.fr
SOURCE: JOURNAL OF PERIODONTOLOGY, (2000 May) 71 (5) 816-24.
Journal code: 8000345. ISSN: 0022-3492.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001012

AB BACKGROUND: Chediak-Higashi syndrome (C-HS) is a rare congenital disease characterized by defective neutrophil function with abnormal lysosomal inclusions, neutropenia, and reduced chemotaxis. The complete syndrome includes oculocutaneous albinism with photophobia, neurologic features, recurrent infections, and enterocolitis. METHODS: A 14-year-old male C-HS patient was referred to us because of serious periodontal destruction with acute inflamed gingiva and ulcers. Clinical and biological investigations were performed, leading to the **diagnosis** of C-HS. RESULTS: Laboratory findings included neutropenia and hypergammaglobulinemia. Peripheral blood smears showed giant granules in neutrophils, eosinophils, and granulocytes. Bone marrow smears showed giant inclusions in leukocyte precursor cells. These granules and inclusions were characteristic of

Chediak-Higashi syndrome. Oral radiographic status showed extensive loss of alveolar bone leading, in most cases, to tooth exfoliation. Bacteria often associated with periodontitis were detected in subgingival plaque samples, including **Fusobacterium** nucleatum, Campylobacter rectus, Prevotella melaninogenica, Peptostreptococcus anaerobius, and Clostridium sp. Biopsies of periodontal tissues for light and electronic microscopic examinations revealed massive bacterial invasion of the epithelial tissue, epithelial cells, and connective tissue. Ultrastructural observations of periodontal polymorphonuclear leukocytes showed defective granulation, with abnormal granules not discharging their lysosomal content against engulfed bacteria. Viable dividing bacteria were found in the cytoplasm. CONCLUSIONS: In this case, early-onset periodontitis seems to be the expression of C-HS granulocyte deficiency. Periodontal treatment of these patients is often unsuccessful. This case report illustrates the importance of the dentist in initiating clinical and biological investigations in such early aggressive periodontitis in young patients.

L31 ANSWER 13 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:107730 BIOSIS
DOCUMENT NUMBER: PREV200000107730
TITLE: Bone and joint infections in the elderly: Practical treatment guidelines.
AUTHOR(S): Mader, Jon T. (1); Shirtliff, Mark E.; Bergquist, Stephen; Calhoun, Jason H.
CORPORATE SOURCE: (1) Section Surgical Infectious Diseases, Division of Infectious Diseases, Department of Internal Medicine, Hyperbaric Facility, Marine Biomedical Institute, University of Texas Medical Branch, New Trauma Building, Galveston, TX, 77555-1115 USA
SOURCE: Drugs & Aging, (Jan., 2000) Vol. 16, No. 1, pp. 67-80. ISSN: 1170-229X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Two types of haematogenous osteomyelitis that are seen in the elderly are vertebral and long bone osteomyelitis. Osteomyelitis secondary to contiguous foci of infection can occur in older adults without vascular insufficiency (secondary to pressure ulcers) or with vascular insufficiency due to diabetes mellitus or peripheral vascular disease from atherosclerosis. Most cases of osteomyelitis can be reasonably treated with adequate drainage, thorough debridement, obliteration of dead space, wound protection, and antimicrobial therapy. Patients are initially given a broad spectrum antimicrobial that is changed to specific antimicrobial therapy based on meticulous bone cultures taken at debridement surgery or from deep bone biopsies. Surgical management is often required in the treatment of osteomyelitis and includes adequate drainage, extensive debridement of all necrotic tissue, obliteration of dead spaces, stabilisation, adequate soft tissue coverage, and restoration of an effective blood supply. Bone repair and bone mineral density may be significantly retarded and may be corrected by eliminating risk factors, supplementing the diet with calcium, bisphosphonates, and/or vitamin D, and treating with testosterone and/or estrogen when deficient. Sodium fluoride treatment and anabolic steroids may be used as alternatives. Septic arthritis is a medical emergency, and prompt recognition and rapid and aggressive treatment are critical to ensuring a good prognosis. The treatment of septic arthritis includes appropriate antimicrobial therapy and joint drainage. Adverse effects of prescribed antibacterials occur more often in the elderly patient than in young adults. The physician can help to minimise the incidence of adverse effects and improve outcomes by

being aware of the principles of clinical pharmacology, the characteristics of specific drugs, and the special physical, psychological and social needs of older patients.

L31 ANSWER 14 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-562048 [47] WPIDS
CROSS REFERENCE: 1999-562050 [47]; 2001-147547 [15]; 2002-750414 [81]
DOC. NO. CPI: C1999-163927
TITLE: New isolated Cytokine receptor Common Gamma Chain Like polypeptides, used for treating, e.g. immune and autoimmune disease.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): MOORE, P A; ROSEN, C A; RUBEN, S M; DUAN, R D; EBNER, R; ENDRESS, G A; FENG, P; KYAW, H; LAFLEUR, D W; NI, J; OLSEN, H S; SHI, Y; SOPPET, D R; WEI, Y; YOUNG, P E; YU, G
PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC; (HUMA-N) HUMAN GENOME SCI; (MOOR-I) MOORE P A; (ROSE-I) ROSEN C A; (RUBE-I) RUBEN S M
COUNTRY COUNT: 84
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9947538	A1	19990923	(199947)*	EN	148
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9930727	A	19991011	(200008)		
EP 1093457	A1	20010425	(200124)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2002506625	W	20020305	(200220)		207
JP 2002506627	W	20020305	(200220)		533
US 2002193305	A1	20021219	(200303)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9947538	A1		WO 1999-US5068	19990305
AU 9930727	A		AU 1999-30727	19990305
EP 1093457	A1		EP 1999-912330	19990305
			WO 1999-US5068	19990305
JP 2002506625	W		WO 1999-US5068	19990305
			JP 2000-536731	19990305
JP 2002506627	W		WO 1999-US5804	19990318
			JP 2000-536733	19990318
US 2002193305	A1	Provisional	US 1998-78563P	19980319
		Provisional	US 1998-86505P	19980522
		CIP of	US 1999-263626	19990305
		CIP of	WO 1999-US5068	19990305
		CIP of	US 1999-376430	19990818
		CIP of	WO 2000-US22493	20000817
		Provisional	US 2001-269876P	20010221
			US 2002-78059	20020220

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9930727	A	Based on	WO 9947538
EP 1093457	A1	Based on	WO 9947538
JP 2002506625	W	Based on	WO 9947538
JP 2002506627	W	Based on	WO 9947540

PRIORITY APPLN. INFO: US 1998-86505P 19980522; US 1998-78563P 19980319; US 1998-78566P 19980319; US 1998-78573P 19980319; US 1998-78574P 19980319; US 1998-78576P 19980319; US 1998-78577P 19980319; US 1998-78578P 19980319; US 1998-78579P 19980319; US 1998-78581P 19980319; US 1998-80312P 19980401; US 1998-80313P 19980401; US 1998-80314P 19980401; US 1999-263626 19990305; US 1999-376430 19990818; WO 2000-US22493 20000817; US 2001-269876P 20010221; US 2002-78059 20020220

AN 1999-562048 [47] WPIDS

CR 1999-562050 [47]; 2001-147547 [15]; 2002-750414 [81]

AB WO 9947538 A UPAB: 20030113

NOVELTY - Isolated Cytokine Receptor Common Gamma Chain Like (CRCGCL) polypeptides are new.

DETAILED DESCRIPTION - (A) A novel isolated nucleic acid molecule (NAM) comprises a polynucleotide (PN) having a nucleotide sequence (NS) at least 95% identical to a sequence selected from:

(a) a PN fragment of sequence (I) of 573 amino acids (aa) or a PN fragment of the cDNA sequence included in ATCC No's 209641 or 209691;

(b) a PN encoding a polypeptide fragment, domain or epitope of sequence (II) of 371 aa or the cDNA sequence included in ATCC No's 209641 or 209691;

(c) a PN encoding a polypeptide of sequence (II) or the cDNA sequence included in ATCC No's 209641 or 209691 having biological activity;

(d) a PN which is an (allelic) variant of sequence (I);

(e) a PN which encodes a species homolog of sequence (II);

(f) a PN capable of hybridizing under stringent conditions to any one of the PNs specified in (a)-(e), where the PN does not hybridize under stringent conditions to a NAM having a NS of only A or T residues.

INDEPENDENT CLAIMS are also included for the following:

(1) a recombinant vector comprising an isolated NAM as in (A);

(2) making a recombinant host cell comprising an isolated NAM as in (A);

(3) a recombinant host cell produced by a method as in (3);

(4) an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from:

(a) a polypeptide fragment, domain or epitope of (II) or the encoded sequence included in ATCC No's 209641 or 209691;

(b) a mature form of or a full length secreted protein, and

(c) a variant, allelic variant or a species homolog of sequence (II);

(5) an isolated antibody that binds specifically to an isolated polypeptide as in (4), and

(6) a recombinant host cell that expresses an isolated polypeptide as in (4).

USE - The novel polypeptide is designated Cytokine Receptor Common Gamma Chain Like (CRCGCL). The tissue distribution in only activated T-cells and homology to the cytokine receptors IL-2 and IL-13 suggests that this protein is a novel member of the cytokine receptor family expressed specifically on T-cells. The tissue distribution of this gene in

cells of the immune system suggests that the protein product of this clone would be useful for treatment, prophylaxis and **diagnosis** of immune and autoimmune diseases, such as lupus, transplant rejection, allergic reactions, arthritis, asthma, immunodeficiency diseases, leukemia, and AIDS. The products can be used in the treatment, prophylaxis and detection of e.g. Graves Disease, lymphocytic thyroiditis, hyperthyroidism, hypothyroidism, Addison's disease, hemolytic anemia, antiphospholipid syndrome, rheumatoid arthritis, dermatitis, allergic encephalomyelitis, glomerulonephritis, Goodpasture's syndrome, Graves' disease, multiple sclerosis, myasthenia gravis, neuritis, ophthalmia, bullous penphigoid, pemphigus, polyendocrinopathies, purpura, Reiter's disease, Stiff-Man syndrome, autoimmune thyroiditis, systemic lupus erythematosus, autoimmune pulmonary inflammation, Guillain-Barre syndrome, insulin dependent diabetes mellitus, autoimmune inflammatory eye disease, allergic reactions and conditions such as asthma (particularly allergic asthma) or other respiratory problems, anaphylaxis, hypersensitivity to an antigenic molecule, blood group incompatibility, organ rejection, graft-versus-host disease, inflammatory conditions, both chronic and acute conditions, including inflammation associated with infection (e.g. septic shock, sepsis, or systemic inflammatory response syndrome), ischemia-reperfusion injury, **endotoxin** lethality, complement-mediated hyperacute rejection, nephritis, cytokine or chemokine induced lung injury, inflammatory bowel disease, Crohn's disease, or resulting from over production of cytokines (e.g. TNF or IL-1), hyperproliferative disorders, e.g. neoplasms, hypergammaglobulinemia, lymphoproliferative disorders, paraproteinemias, purpura, sarcoidosis, Sezary syndrome, Waldenstron's macroglobulinemia, Gaucher's disease, histiocytosis, infectious agents, e.g. virus, bacterial, fungal or parasitic infections. They can also be used to differentiate, proliferate and attract cells, leading to the regeneration of tissues e.g. to repair, replace, or protect tissue damaged by congenital defects, trauma (wounds, burns, incisions, or ulcers), age, disease (e.g. **osteoporosis**, osteocarthritis, periodontal disease, liver failure), surgery, including cosmetic plastic surgery, fibrosis, reperfusion injury or systemic cytokine damage. They can also be used to treat e.g. blood protein disorders (e.g. agammaglobulinemia, dysgammaglobulinemia), ataxia telangiectasia, common variable immunodeficiency, Digeorge Syndrome, HIV infection, HTLV-BLV infection, leukocyte adhesion deficiency syndrom e, lymphopenia, phagocyte bactericidal dysfunction, severe combined immunodeficiency, Wiskott-Aldrich disorder, anemia, thrombocytopenia, or hemoglobunura, to modulate hemostatic or thrombolytic activity, e.g. to treat blood coagulation disorders such as afibrinogenemia, or factor deficiencies, blood platelet disorders (e.g. thrombocytopenia), or wounds resulting from trauma, surgery or other causes, or to decrease hemostatic or thrombolytic activity to inhibit or dissolve clotting, important in the treatment of heart attacks, strokes or scarring. The products can also increase or decrease the differentiation or proliferation of embryonic stem cells and hematopoietic lineage, to modulate mammalian characteristics, such as body weight, height, hair, weight, hair color, eye color, skin, percentage of adipose tissue, pigmentation, size, and shape, (e.g. cosmetic surgery), to modulate mammalian metabolism affecting catabolism, anabolism, processing utilization and storage of energy, to change a mammal's mental state or physical state by influencing biorhythms, caricadic rhythms, depression (including depressive disorders), tendency for violence, tolerance for pain, reproductive capabilities (preferably by Activin or Inhibin-like activity), hormonal or endocrine levels, appetite, libido, memory, stress, or other cognitive qualities, used as a food additive or preservative, such as to increase or decrease storage capabilities, fat content, lipid, protein, carbohydrate, vitamins, minerals, cofactors or other nutritional components. The

products can also be used for detection and the production of transgenic animals.

ADVANTAGE - None given.

Dwg.0/3

L31 ANSWER 15 OF 28 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2000-061922 [05] WPIDS
CROSS REFERENCE: 2000-572072 [53]; 2002-673824 [72]; 2003-352213 [33]; 2003-352290 [33]
DOC. NO. NON-CPI: N2000-048546
DOC. NO. CPI: C2000-017067
TITLE: New tumor necrosis factor receptor-like polypeptides used to, e.g. treat Digeorge syndrome.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): NI, J; RUBEN, S M
PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9920758	A1	19990429	(200005)*	EN	166
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9911023	A	19990510	(200005)		
EP 1025228	A1	20000809	(200039)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2001520039 W		20011030	(200202)		216

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9920758	A1	WO 1998-US22085	19981021
AU 9911023	A	AU 1999-11023	19981021
EP 1025228	A1	EP 1998-953724	19981021
		WO 1998-US22085	19981021
JP 2001520039 W		WO 1998-US22085	19981021
		JP 2000-517079	19981021

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9911023	A	Based on	WO 9920758
EP 1025228	A1	Based on	WO 9920758
JP 2001520039 W		Based on	WO 9920758

PRIORITY APPLN. INFO: US 1997-63212P 19971021
AN 2000-061922 [05] WPIDS
CR 2000-572072 [53]; 2002-673824 [72]; 2003-352213 [33]; 2003-352290 [33]
AB WO 9920758 A UPAB: 20030526

NOVELTY - New tumor necrosis factor receptor-like polypeptides (P1)-(P3) designated TR11, TR11SV1 and TRIISV1 are used to treat disorders associated with abnormal cell survival.

DETAILED DESCRIPTION - (A) A nucleic acid molecule (NAM) has

polynucleotide (PN) with nucleotide (nt) sequence (NS) at least 95% identical to:

(a) NS encoding P1 having complete amino acid (aa) sequence (CAS) (aa -25 to 209 in sequence (I) of 234 aa);

(b) NS encoding P2 having CAS (aa 1-241 in sequence (II) of 241 aa);

(c) NS encoding P3 having CAS (aa -19 to 221 in sequence (III) of 240 aa);

(d) NS encoding CAS as in (a)-(c), but in all lacking the N-terminal methionine, i.e. aa -24 to 209 in (I), 2 to 240 in (II) and -18 to 221 in (III), respectively;

(e) NS encoding predicted mature TR11 receptor (R1) having an aa sequence at positions 26-234 (aa 1-209 in (I));

(f) NS encoding predicted mature TR11SV2 receptor (R3) having an aa sequence at positions 20-240 (aa 1-221 in (III));

(g) NS's encoding P1-P3 having CAS encoded by a cDNA clone contained in ATTCC No. 209340, ATCC No. 209341 and ATCC No. 209342, respectively ;

(h) NS's encoding a mature R1, TR11SV1 (R2) and R3 having aa sequences encoded by a cDNA clone as in (g);

(i) NS's encoding R1-R3 extracellular domains (ED's);

(j) NS's encoding R1-R3 transmembrane domains (TD's);

(k) NS's encoding R1-R3 intracellular domains (ID's);

(l) NS's encoding R1-R3 ED's and ID's with all or part of TD's deleted,

(m) complementary to any of (a)-(l) (all sequences given in the specification).

INDEPENDENT CLAIMS are also included for the following:

(1) NAM comprising PN hybridizing to a PN having a NS as in (Aa)-(Al) or (m), where the PN which hybridizes does not hybridize to PN having NS having only A or T residues;

(2) NAM comprising PN encoding aa sequence of an epitope-bearing portion of R1-R3 having a sequence as in (Aa)-(Al) or (m);

(3) NAM comprising PN having sequence:

(a) NS of fragment of sequence (V) of 983 nt, comprising at least 30-50 contiguous nt from (I), provided that the NAM is not (VI) and (VII) of 466 and 581 nt, or any subfragment;

(b) NS of fragment of sequence (VIII) of 1007 nt comprising at least 30-50 contiguous nt from (VIII), provided that the NAM is not (VI), (VII) or any subfragment;

(c) NS of a fragment of sequence (IX) of 1074 nt comprising at least 30-50 contiguous nt from (IX), provided that the isolated NAM is not (VI), (VII) or any subfragment, and

(d) NS complementary to (a), (b) or (c);

(4) making a recombinant vector (RV) by inserting NAM as in (A) into it;

(5) RV produced by (4);

(6) making a recombinant host cell (RHC) by introducing RV of (5) into it;

(7) RHC produced by (6);

(8) a polypeptide having an aa sequence at least 95% identical to:

(a) P1 encoded by the deposited cDNA:

(i) including the leader, or

(ii) minus the leader, (i.e. the mature protein);

(b) P1 of (I):

(i) as in (8a), or

(ii) including the leader but minus the N-terminal methionine;

(c) a polypeptide of (I) minus the leader;

(d) an ED, TD and ID of R1 of (I);

(e) a complete or mature P2 encoded by the deposited cDNA;

(f) P2 of (II);

(g) P2 of (II) including the leader but minus the N-terminal

methionine;
(h) ED of R2 of (II);
(i) P3 encoded by the deposited cDNA including the leader;
(j) P3 encoded by a deposited cDNA:
(i) minus the leader (i.e. the mature protein); or
(ii) including the leader;
(k) P3 of (II);
(l) P3 of (II) including the leader but minus the N-terminal methionine;
(m) polypeptide of (III) minus the leader, and
(n) ED of R3 of (III), and
(9) an antibody specific to a polypeptide as in (8) (all sequences are given in the specification).

USE - P1-P3 are novel members of the tumor necrosis factor (TNF) receptor family. The polypeptides may be involved in the regulation of cell-type specific receptor-mediated cell growth, differentiation, and ultimately, cell death. They can be used for screening for agonists/antagonists (claimed). The polypeptides, agonists or antagonists can be used for treating a disease state associated with aberrant cell survival (claimed). They can be used for treating immune deficiency disorders, e.g. blood protein disorders (e.g. agammaglobulinemia, dysgammaglobulinemia), ataxia teangiectasia, common variable immunodeficiency, Digeorge syndrome, HIV infection, HTLV-BLV infection, leukocyte adhesion deficiency syndrome, lymphopenia, phagocyte bactericidal dysfunction, severe combined immunodeficiency (SCIDs), Wiskott-Aldrich disorder,anemia, thrombocytopenia, hemoglobinuria, blood coagulation disorders (e.g. afibinogenemia, factor deficiencies), blood platelet disorders (e.g. thrombocytopenia), or wounds resulting from trauma, surgery, or other causes, to treat heart attacks (infarction), stokes or scarring, Addison's disease, hemolytic anemia, antiphospholipid syndrome, rheumatoid arthritis, dermatitis, allergic encephalomyelitis, glomerulonephritis, Goodpastures syndrome, Grave's disease, multiple sclerosis, myasthenia gravis, neuritis, ophthalmia, bullous pemphigoid, pemphigus, polyendocrinopathies, purpura, Reiter's disease, Stiff-Man syndrome, autoimmune thyroiditis, systemic lupus erythematosus, autoimmune pulmonary inflammation, Guillain-Barre syndrome, insulin dependent diabetes mellitus or autoimmune inflammatory eye disease, anaphylaxis, hypersensitivity to an antigenic molecule or blood group incompatibility, organ rejection or graft versus host disease, inflammatory conditions, both chronic and acute conditions, including inflammation associated with infection (e.g. septic shock, sepsis or systemic inflammatory response syndrome (SIRS)), iscemia-reperfusion injury, endotoxin lethality, arthritis, complement-mediated hyperacute rejection, nephritis, cytokine or chemokine induced lung injury, inflammatory bowel disease, Crohn's disease, or resulting from overproduction of cytokines (e.g. TNF or IL-1), hyperproliferative disorders, e.g. neoplasms, hypergammaglobulinemia, lymphoproliferative disorders, paraproteinemias, pupura, sarcoidosis, Sezary sundrome, Waldenstron's macroglobulinemia, Gaucher's disease, histiocytosis or infections caused by viruses, bacteria, fungi or parasites, rheumatoid arthritis, osteoarthritis, psoriasis, septicemia or inflammatory bowel disease. They can also be used to repair, replace, or protect tissue damaged by congenital defects, trauma (wounds, burns, incisions, or ulcers), age, disease (e.g. osteoporosis, osteocarthritis, periodontal disease, liver failure), surgery, including cosmetic plastic surgery, fibrosis, reperfusion injury or systemic cytokin e damage, peripheral nerve injuries, peripheral neuropathy (e.g. resulting from chemotherapy or other medical therapies), localized neuropathies, and central nervous system disease (e.g. Alzheimer's disease, Parkinson's disease, Huntingtons disease, amyotrophic lateral sclerosis, and Shy-Drager syndrome). The products can also be used for detection,

diagnosis and prognosis.
ADVANTAGE - None given.l
Dwg.0/7

L31 ANSWER 16 OF 28 MEDLINE on STN
ACCESSION NUMBER: 1999360401 MEDLINE
DOCUMENT NUMBER: 99360401 PubMed ID: 10433019
TITLE: Severe complications of ulcerative colitis after high-dose prednisolone and azathioprine treatment.
AUTHOR: Matsuda K; Watanabe T; Abo Y; Uchida H; Kawamura Y J; Masaki T; Muto T
CORPORATE SOURCE: The Department of Surgery, The University of Tokyo, Japan.
SOURCE: JOURNAL OF GASTROENTEROLOGY, (1999 Jun) 34 (3) 390-4. Journal code: 9430794. ISSN: 0944-1174.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19990925
Last Updated on STN: 19990925
Entered Medline: 19990914

AB We report a rare case of ulcerative colitis (UC) associated with methicillin-resistant **Staphylococcus aureus** (MRSA) and Pseudomonas aeruginosa infections in multiple organs, and with compressive fracture from **osteoporosis** after the administration of high-dose prednisolone and azathioprine. A 25-year-old man had been treated with high-dose prednisolone for UC. He suddenly experienced severe lumbago, which prevented him from walking. Plain X-ray demonstrated compressive fractures of the thoracic and the lumbar vertebrae, which were thought to be due to **osteoporosis** as a side effect of the high-dose prednisolone. At this admission, in another hospital, he also had a bloody discharge from the rectum, and azathioprine was started; however, the patient's condition still did not show any improvement. The total doses of azathioprine and prednisolone he had received were 3150 mg and more than 15,000 mg, respectively. Considering the presence of the serious complications, surgical intervention was the treatment selected. Culture study revealed MRSA in the feces and nasal cavity, and P. aeruginosa in the feces and urine. Vancomycin hydrochloride and gentamicin were administered, and were effective, with a subsequent negative culture study. Subtotal colectomy with mucus fistula was performed. After the operation, culture studies remained negative. Major steroid side effects such as bone fracture and **osteoporosis** should be considered as an indication for surgery in UC patients. MRSA and P. aeruginosa are a menace, especially for UC immunosuppressed patients on steroid or immunosuppressive therapy. When these bacteria are detected, there should be prompt and adequate antimicrobial therapy against the organisms and the immunosuppressive therapy should be immediately discontinued. We conclude that surgical therapy should be considered in the earlier stage for patients with intractable UC, rather than continuing long-term administration of steroid or azathioprine, which may lead to serious complications.

L31 ANSWER 17 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 1998092750 EMBASE
TITLE: A monthly critical overview of current medicine.
AUTHOR: Stollerman G.H.; Bisno A.L.
CORPORATE SOURCE: Dr. G.H. Stollerman, 30 Rutgers Road, Wellesley, MA 02181, United States
SOURCE: Hospital Practice, (15 Feb 1998) 33/2 (71-74).

ISSN: 8750-2836 CODEN: HOPRBW
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
010 Obstetrics and Gynecology
037 Drug Literature Index
LANGUAGE: English

L31 ANSWER 18 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 97203346 EMBASE
DOCUMENT NUMBER: 1997203346
TITLE: Therapy of atopic dermatitis.
AUTHOR: Cerio R.
CORPORATE SOURCE: R. Cerio, Department of Dermatology, Royal Hopitals NHS Trust, Royal London Hospital, Whitechapel, London El 1BB, United Kingdom
SOURCE: Journal of the European Academy of Dermatology and Venereology, (1997) 8/SUPPL. 1 (S6-S10).
Refs: 12
ISSN: 0926-9959 CODEN: JEAVEQ
PUBLISHER IDENT.: S 0926-9959(97)00054-8
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background: Appropriate management of patients with atopic dermatitis must depend primarily on early, accurate **diagnosis** and allergen avoidance in infants. Patients with less typical lesions, such as extensor involvement, may be misdiagnosed. Aim: Dermatologists should try to ensure that correct management is initiated in the primary care setting and maintained by patients and parents. Guidelines for the management of both paediatric and adult patients with atopic dermatitis have been drawn up by an international panel of dermatologists. For patients with mild disease, elaborate investigations to identify the trigger factors are unnecessary. Management should include general measures (skin hydration, irritant and allergen avoidance), provision of information to parents and intermittent use of low potency topical corticosteroids when necessary. For patients with mild to moderate disease, investigations may require assessment of food allergies in paediatric cases and of contact allergens and irritants in adult cases. Identification of **Staphylococcus aureus** and herpes simplex virus secondary infection is also important. Conclusions: General measures remain important in mild to moderate atopic dermatitis, with appropriate education and support for parents. Topical corticosteroids play an important role, possibly with antihistamines to help alleviate the pruritus. In certain situations, systemic treatment with antibiotics, corticosteroids and other agents may be indicated.

L31 ANSWER 19 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 95336652 EMBASE
DOCUMENT NUMBER: 1995336652
TITLE: Fracture of the iliac crest following bone grafting: A case report and literature review.
AUTHOR: Friend K.D.; Koval K.J.; Mirovsky Y.; Remer S.S.; Bloom N.; Neuwirth M.G.
CORPORATE SOURCE: Hospital for Joint Diseases, 301 East 17th Street,New York,

NY 10003, United States
SOURCE: Bulletin: Hospital for Joint Diseases, (1995) 54/1 (49-51).
ISSN: 0018-5647 CODEN: BHJDEI
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
033 Orthopedic Surgery
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Fracture of the anterior iliac crest following bone grafting is an extremely rare occurrence. Five cases have been reported in the literature, none of which were internally stabilized. We are reporting a sixth case. Of the six cases, our harvest site is the furthest posterior from the anterior superior iliac spine. The fracture resulted in a large displaced anterior fragment that required open reduction and internal fixation with plates and screws. **Osteoporosis** increases the risk of anterior iliac crest fractures following bone grafting, but preventive procedures can be performed.

L31 ANSWER 20 OF 28 MEDLINE on STN
ACCESSION NUMBER: 92035882 MEDLINE
DOCUMENT NUMBER: 92035882 PubMed ID: 1934731
TITLE: Resection arthroplasty for septic arthritis of the hip in ambulatory and nonambulatory adult patients.
AUTHOR: Milgram J W; Rana N A
CORPORATE SOURCE: Department of Orthopaedic Surgery, Northwestern University Medical School, Chicago, Illinois.
SOURCE: CLINICAL ORTHOPAEDICS AND RELATED RESEARCH, (1991 Nov) (272) 181-91.
Journal code: 0075674. ISSN: 0009-921X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199112
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19970203
Entered Medline: 19911223
AB Twenty-three patients who required resection arthroplasty because of pyogenic infection of a hip joint were reviewed. Nine of the patients were ambulatory, and the others were either paraplegic or bed-ridden because of chronic neurologic disease. The average duration of symptoms was more than two months. The only consistently abnormal laboratory test was the erythrocyte sedimentation rate (ESR). Joint-space narrowing and bone erosion due to osteomyelitis were the most common roentgenographic findings. Different microorganisms were isolated from the different cases, but **Staphylococcus aureus** was documented in eight hips. Femoral head dislocation of subluxation was documented in 11 of 24 hips. Osteoarthrosis or osteonecrosis was a preexisting condition in only four hips, all in ambulatory patients. Pathologic findings included loss of articular cartilage by surface erosion and by subchondral bone resorption, resulting in the separation of the cartilage from the underlying bone, bone erosion, osteomyelitis, and segmental osteonecrosis.

L31 ANSWER 21 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 91234593 EMBASE
DOCUMENT NUMBER: 1991234593
TITLE: Cytokine production by peripheral blood cells in postmenopausal **osteoporosis**.

AUTHOR: Zarrabeitia M.T.; Riancho J.A.; Amado J.A.; Napal J.; Gonzalez-Macias J.
CORPORATE SOURCE: Departamento de Medicina Interna, Hospital M. Valdecilla, Universidad de Cantabria, Santander, Spain
SOURCE: Bone and Mineral, (1991) 14/2 (161-167).
ISSN: 0169-6009 CODEN: BOMIET
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
025 Hematology
026 Immunology, Serology and Transplantation
033 Orthopedic Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English

AB It has been suggested that the release of cytokines with bone-resorbing activity from cells of the immune system might have a role in the pathogenesis of **osteoporosis**. We measured the secretion of the bone-resorbing products tumor necrosis lactor, interleukin 1.beta. and PGE2 by peripheral blood mononuclear cells from seven healthy postmenopausal women and 12 patients with postmenopausal **osteoporosis**. No differences were observed between both groups either in unstimulated cultures or in cultures activated with calcitriol, **endotoxin** or phorbol esters. These results give no support for a role of peripheral blood immune cells in postmenopausal **bone loss**.

L31 ANSWER 22 OF 28 JICST-EPlus COPYRIGHT 2003 JST on STN
ACCESSION NUMBER: 910295737 JICST-EPlus
TITLE: Immunological **diagnosis** of advanced periodontitis.
AUTHOR: ISHIKAWA ISAO
CORPORATE SOURCE: Tokyo Medical and Dental Univ., Faculty of Dentistry
SOURCE: Nippon Shika Igakkaishi (Journal of the Japanese Association for Dental Science), (1991) vol. 10, pp. 118-123. Journal Code: Y0002A (Fig. 7, Tbl. 2, Ref. 5)
ISSN: 0286-164X
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

AB Serum IgG antibodies to seven periodontopathic bacteria were assessed with an enzyme-linked immunosorbent assay(ELISA) in 56 patients with various types of periodontitis and in 26 control with healthy periodontum. The severity of **bone loss** were measured on the radiographs of the patients. Bacteroides gingivalis, B. loescheii, **Fusobacterium** nucleatum, Actinobacillus actinomycetemcomitans, Eikenella Corrodens, B. intermedius and Caponocytophaga ochracea were the bacterial strains of interest. Antigens were prepared by cold untrasonication of washed bacterial cells. Association of high- or low-IgG antibody titer to the bacteria was evaluated. High or low titer of IgG were based on ELISA measurements in healthy subjects. Values exceeding upper or lower two times of the standard deviation were classified with high or low titers. Most patients (76.8%) with various types of periodontitis exhibited high-IgG antibody titers against one or some of the bacteria examined. B. gingivalis was predominantly associated with periodontitis patients (60.7%). A actinomycetemcomitans was found in a few patients (12.5%), who showed a severe and more rapid **bone loss**. In this paper, three representative periodontal cases were presented with the ELISA results. ELISA measurements reinforce the traditional periodontal **diagnosis** methods to determine the

treatment planning. (author abst.)

L31 ANSWER 23 OF 28 MEDLINE on STN
ACCESSION NUMBER: 91162449 MEDLINE
DOCUMENT NUMBER: 91162449 PubMed ID: 2002433
TITLE: Microbiological and immunological aspects of experimental periodontal disease in rats: a review article.
AUTHOR: Klausen B
CORPORATE SOURCE: Department of Microbiology, Royal Dental College, Copenhagen, Denmark.
SOURCE: JOURNAL OF PERIODONTOLOGY, (1991 Jan) 62 (1) 59-73. Ref: 142
Journal code: 8000345. ISSN: 0022-3492.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910505
Last Updated on STN: 19910505
Entered Medline: 19910412

AB Animal models in which microbiological and immunological aspects of periodontal disease can be studied prospectively seem well warranted. The rat bears much resemblance to man with respect to periodontal anatomy, development and composition of dental plaque, histopathology of periodontal lesions, and basic immunobiology. Furthermore, reproducible methods are available for assessment of periodontal disease in rats, and detectable periodontal destruction can be induced in a few weeks in these animals without traumatizing periodontal tissues with ligatures. Experimental periodontitis studies in germ-free rats have confirmed the pathogenicity of several suspected periodontal pathogens (Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Capnocytophaga sputigena, Eikenella corrodens, and **Fusobacterium** nucleatum). The studies also suggest that the number of periodontal pathogens may be higher than generally believed, since species like Streptococcus sobrinus and Actinomyces viscosus are associated with periodontal **bone loss** in rats. Studies in rats with congenital or induced immune defects indicate that generalized or selective immunosuppression at the time of infection with periodontal pathogens may aggravate periodontal disease. Studies in immunized rats indicate that periodontal disease can be prevented by immunization against periodontal pathogens. However, it is also possible by immunization to induce periodontal destruction; i.e., the immune system has a destructive potential which should not be overlooked. In the future, the rat model may prove valuable for initial **screening** of antigen preparations and immunization regimens in the search for a periodontitis vaccine.

L31 ANSWER 24 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 91154442 EMBASE
DOCUMENT NUMBER: 1991154442
TITLE: Virulence factors of oral bacteroides.
AUTHOR: Mouton C.
CORPORATE SOURCE: Groupe de Recherche en Ecologie Buccale, Ecole de Medicine Dentaire, Universite Laval, Quebec, Que., Canada
SOURCE: Medecine et Maladies Infectieuses, (1990) 20/SPEC. ISS. DEC. (153-164).
ISSN: 0399-077X CODEN: MMAIB5
COUNTRY: France

DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 004 Microbiology
011 Otorhinolaryngology
LANGUAGE: French
SUMMARY LANGUAGE: English

AB Periodontal diseases result in the destruction of tooth supporting tissues eventually leading, if untreated, to tooth loss. The most common form, chronic adult periodontitis (AP), is prevalent in humans after the fourth decade of life. It has recently been reported that 77% of adults in the USA had at least one site with periodontal attachment loss of 2 mm or more, and that, on average, 6 sites were affected. It is now well established that bacteria play a central role in the initiation and progression of these inflammatory diseases. Because of the complexity of the oral flora (about 200 taxa have been found in periodontal pockets), precise information about the etiologic bacteria is difficult to ascertain. However, the role of specific groups of microorganisms, including Gram-negative anaerobic bacteria, in the pathogenesis of periodontial diseases has been increasingly appreciated in recent years, so that these are nowadays considered polymicrobial, mixed, infections. Studies of the predominant cultivable microbiota have revealed that black-pigmented Bacteroides spp (BPB) are frequently associated with active destructive periodontal lesions of AP together with Wolinella recta, **Fusobacterium** nucleatum, Eubacterium spp., and Bacteroides forsythus, to name a few. Actinobacillus actinomycetemcomitans, an aerotolerant, capnophilic Gram-negative species, has quite specifically been implicated in the etiology of the juvenile and localized form of periodontitis. The taxonomy or oral Bacteroides spp has undergone substantial change in the last decade. Eight species of oral black-pigmented Bacteroides and 11 species of non-pigmented oral Bacteroides are now recognized. It is customary to distinguish asaccharolytic from saccharolytic species with in the group of black-pigmented Bacteroides. Of the saccharolytic species 5 are isolated from the oral cavity of man or animals: B. melaninogenicus, B. denticola, B. loescheii, B. intermedius and B. macacae, and 2 are extra-oral: B. corporis and B. levii. Recently, it has been proposed to reclassify the three asaccharolytic black-pigmented Bacteroides spp, B. gingivalis, B. endodontalis and B. asaccharolyticus, to the new genus Porphyromonas. All three organisms have been associated with human diseases: B. gingivalis in periodontal diseases; B. endodontalis in dental root canal infections; and B. asaccharolyticus in pelvic and bite wound infections, and endometritis. The primary habitat of B. gingivalis is the dento-gingival area; B. endodontalis is seldom found in the gingival sulcus, and B. asaccharolyticus is almost never found in the oral cavity. Thus, it appears that each of these species occupies a unique ecological niche in the human body. A recent experiment resulted in the successful implantation of a rifampicinresistant B. gingivalis into the periodontal microbiota of the macaque monkey. An increase in the systemic level of antibody to the microorganism and a burst of **bone loss** were observed, indicating that the emergence of B. gingivalis in the subgingival microflora is capable of inducing periodontitis. Most of the oral BPBs are not infectious for mice or guinea pigs when injected as pure cultures. However, virulent strains of B. gingivalis have been reported which caused abcesses or were invasive upon subcutaneous injection. Yet it is not clear whether B. gingivalis strains recovered from active destructive periodontal lesions are always infectious or invasive in animal models. The periodontopathic potential of BPBs, in particular of B. gingivalis, is documented mostly by studies of the factors which may contribute to the virulence of these bacteria. It must be noted that no exotoxin has been described in this species. Factors important for colonization, in terms of adherence, include fimbriae and extra-cellular

vesicules.

L31 ANSWER 25 OF 28 JICST-EPlus COPYRIGHT 2003 JST on STN
ACCESSION NUMBER: 890429897 JICST-EPlus
TITLE: Epidemiological and microbiological surveys of juvenile periodontitis in Japanese high school students.
AUTHOR: YOSHIDA ICHIE; FUJIWARA TAKU; OOSHIMA TAKASHI; SOBUE SHIZUO
CORPORATE SOURCE: Osaka Univ., Dental School
SOURCE: Shoni Shikagaku Zasshi (Japanese Journal of Pediatric Dentistry), (1988) vol. 26, no. 4, pp. 782-789. Journal Code: Y0025A (Fig. 6, Tbl. 1, Ref. 23) ISSN: 0583-1199
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New
AB The prevalence of juvenile periodontitis was examined in 3603 high school students by using a three-stage diagnostic procedure. The subjects were screened initially by assessment of the mobility of the upper central incisors. The number of positive subjects came to 38 males and 22 females, followed by assessment of probing depths of the pockets around the central incisors and first molars. The number of positive subjects with more than 4mm of pocket depths came to 7 males and 4 females. Subsequently, a full clinical and radiographic examination was performed on 5 of them, and one female subject was diagnosed as incipient juvenile periodontitis(0.03% of the total subjects). The patient enjoyed good general health. Radiographic examination showed vertical **bone loss** around central incisors and left first molars. Subgingival plaques were taken from the patients under the anaerobic conditions, and the subgingival flora was composed of 51% obligative anaerobic, 17% facultative anaerobic, 32% unknown bacteria, respectively. Predominant bacteria were **Fusobacterium** and black-pigmented Bacteroides. Actinobacillus actinomycetemcomitans was also isolated. After treatment with subgingival scaling and root planing for 6 months, the predominant bacteria in the subgingival flora was found to shift from Gram negative rods to Gram positive rod Actinomyces, which are usually associated with from healthy subjects.(author abst.)

L31 ANSWER 26 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 82002095 EMBASE
DOCUMENT NUMBER: 1982002095
TITLE: **Endotoxin** inhibition of macrophage-mediated bone resorption.
AUTHOR: Kahn A.J.; Teitelbaum S.L.
CORPORATE SOURCE: Div. Bone Mineral Metab., Washington Univ. Sch. Med., St Louis, MO 63110, United States
SOURCE: Calcified Tissue International, (1981) 33/3 (269-275). CODEN: CATRBZ
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
033 Orthopedic Surgery
LANGUAGE: English
AB The mechanisms by which bacterial **endotoxins** (ETX) elicit **bone loss** in septic osteolytic lesions and in organ cultures of bone rudiments have never been clearly established. The possible mechanisms for ETX action include: (a) the stimulation of osteoclast proliferation; (b) the stimulation of synthesis of secondary agents known to elicit bone resorption, e.g. prostaglandins; (c) the

stimulation of the resorptive activity of osteoclasts. In the absence of extant methods for isolating and culturing osteoclasts, we have explored the last possibility by evaluating the action of ETX in a bone resorption system consisting of a putative osteoclast precursor, the macrophage, cocultured with topically lalebed devitalized bone. We have observed the following: ETX from several species of bacteria (Escherichia coli, Shigella flexneri, and S. minnesota suppress bone resorption (i.e., 45Ca release) mediated by thioglycollate-elicited peritoneal macrophages. This inhibition occurs at ETX concentrations as low as 0.5 .mu.g/ml and is evident within the initial 24 h of incubation. In marked contrast, ETX does not alter the resorptive activity of resident peritoneal macrophages. The suppression of bone resorption by ETX does not depend on the presence of serum complement nor is it a manifestation of reduced cell viability or cell bone-particle binding. Moreover, prolonged pretreatment of elicited cells with ETX does not reduce their subsequent resorptive activity. The suppressive action of ETX is partially reversed by polymyxin B, an observation which implicates the lipid A component of ETX in the inhibitory process. PGE1, PGE2, and indomethacin at concentrations as high as 10-5M do not alter macrophage-mediated resorption; neither does indomethacin modify the action of ETX when the two agents are used concurrently. However, PGE1 and PGE2 can mitigate the suppressive action of ETX. The latter result indicates but does not define a role for prostaglandin in the ETX phenomenon. We suggest the ETX elicits **bone loss** in vivo by stimulating osteoclast proliferation or prostaglandin synthesis, and not by directly evoking enhanced bone resorption by osteoclasts or other osteolytic cells.

L31 ANSWER 27 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 80035351 EMBASE
DOCUMENT NUMBER: 1980035351
TITLE: A study of the bacteria associated with advancing periodontitis in man.
AUTHOR: Tanner A.C.R.; Haffer C.; Bratthall G.T.; et al.
CORPORATE SOURCE: Forsyth Dent. Cent., Boston, Mass., United States
SOURCE: Journal of Clinical Periodontology, (1979) 6/5 (278-307). CODEN: JCPEDZ
COUNTRY: Denmark
DOCUMENT TYPE: Journal
FILE SEGMENT: 011 Otorhinolaryngology
004 Microbiology
LANGUAGE: English

AB Samples of apical plaque were taken by means of an anaerobic gas-flushed syringe from 21 sites in eight patients. The samples were anaerobically dispersed, diluted and plated and incubated in an atmosphere of 80% N2, 10% H2 and 10% CO2 for 7-21 days. All colonies on plates containing 20-50 isolates were picked, repeatedly restreaked, characterized and identified where possible by a probabilistic computer identification program. The sites were divided into four groups on the basis of clinical features. The significance of differences between bacterial populations in the groups was determined by the Kruskal Wallis and Mann-Whitney U tests, while the Spearman rank correlation coefficient was used to determine the rank correlation of clinical features of disease and microbial species. The subgingival microbiota in advanced destructive sites was predominated by Gram-negative rods. The microbiota of two young adult patients with generalized extensive **bone loss**, extensive clinical inflammation and suppuration was dominated by Bacteroides asaccharolyticus and an organism with characteristics consistent with Actinobacillus actinomycetemcomitans. The predominant cultivable microbiota in two patients with extensive **bone loss** but minimal clinical inflammation was predominated by Bacteroides melaminogenicus ss

intermedius and Eikenella corrodens in one patient and E. corrodens and a slow growing fusiform-shaped Bacteriodes in a second patient. A third group of four patients demonstrated moderate levels of clinical inflammation and evidence of continued **bone loss** in the last year. Predominant organisms in this group were more heterogeneous and included B. asaccharolyticus, **Fusobacterium** nucleatum, the 'fusiform' Bacteroides and anaerobic vibrios. Sites with minimal disease in the patients revealed higher proportions of Gram-positive organisms including Rothia Dentocariosa, Actinomyces naeslundii and Actinomyces viscosus. A positive rank correlation could be detected between clinical inflammation including suppuration and B. asaccharolyticus and a negative rank correlation between inflammation and E. corrodens.

L31 ANSWER 28 OF 28 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 76154109 EMBASE
DOCUMENT NUMBER: 1976154109
TITLE: Transactions of the American Clinical and Climatological Association..
AUTHOR: Gordon Jr. D.C.
CORPORATE SOURCE: Baltimore City Hosp., Baltimore, Md., United States
SOURCE: (1975) (234p.).
DOCUMENT TYPE: Book
FILE SEGMENT: 006 Internal Medicine
LANGUAGE: English
AB The proceedings of the 87th meeting of this association are reported. An extensive report of the private meeting is presented including all the lectures, a literature list and discussion. Various different aspects of internal medicine are dealt with like the possible etiological mechanism of tropical sprue, the illnesses of Harry Hopkins, advisor to Presient Roosevelt, and of Sir William Osler; a computer program for **diagnosis** in hematology; attempts to measure quality of health; participation by internists in primary care; antigenic differences among pyocins of Pseudomonas aeruginosa; nature of exotoxin tolerance; different aspects of connective tissue disease; pickwickian syndrome; bone density in normal population and **osteoporosis**; some aspects of the renin angiotensin system; primary aldosteronism and malignant adrenocortical carcinoma; determinants of diuretic responsiveness; selectivity of autonomic control of the peripheral circulation in man; reactive hyperemia as an index to coronary artery narrowing; relation of precapillary pulmonary hypertension to pulmonary venous hypertension and management of patients with WPW syndrome.

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=> d his ful

     FILE 'HCAPLUS' ENTERED AT 17:22:21 ON 22 AUG 2003
L10        13791 SEA ABB=ON  ?OSTEOPOROS? OR ?BONE?(W)?LOSS?
L11           11 SEA ABB=ON  L10 AND ?PROTEIN?(W)?ASSAY?
L12         1110 SEA ABB=ON  L10 AND (?SCREENING? OR ?DIAGNOSIS?)
L13          557 SEA ABB=ON  L10 AND ?SCREENING?
L14            0 SEA ABB=ON  L13 AND ?ENDOTOXIN?(W)(LPS? OR ?LIPOPOLYSACCHARIDE?
                   )
L15            2 SEA ABB=ON  L13 AND ?ENDOTOXIN?
L16            0 SEA ABB=ON  L13 AND ?GAPSTATIN?
L17            0 SEA ABB=ON  L13 AND (?DERMONECROTIC?(W)?TOXIN? OR DNT)
L18            0 SEA ABB=ON  L12 AND ?ENDOTOXIN?(W)(LPS? OR ?LIPOPOLYSACCHARIDE?
                   )
L19            3 SEA ABB=ON  L12 AND ?ENDOTOXIN?
L20            1 SEA ABB=ON  L12 AND ?GAPSTATIN?
L21            1 SEA ABB=ON  L12 AND ?DERMONECROT?
L22            0 SEA ABB=ON  L19 AND L20 AND L21
L23            0 SEA ABB=ON  L15 AND L20 AND L21
L24            4 SEA ABB=ON  L15 OR L19 OR L20 OR L21
L25            1 SEA ABB=ON  L12 AND (S OR ?STAPHYLOCOCCUS?)(W)?AUREUS?
L26            1 SEA ABB=ON  L12 AND ?BRONCHISEPTICA?
L27            1 SEA ABB=ON  L12 AND ?FUSOBACTERIUM?
L28            1 SEA ABB=ON  L25 OR L26 OR L27
L29            4 SEA ABB=ON  L24 OR L28
     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     17:29:51 ON 22 AUG 2003
L30           28 SEA ABB=ON  L29
L31           28 DUP REMOV L30 (0 DUPLICATES REMOVED)
```

0 hits for all 3 "host. produced factors"

4 hits of those combined with OR

1 hit for "any one" of 3 bacteria types

4 hits for any of 6 items listed, i.e., "host. produced factors" or bacterial

28 hits from other databases